STIC-EIC1600/2900 273977

From: NELSON BLAKELY III [nelson.blakelyiii@uspto.gov]
Sent: Wednesday, October 01, 2008 10:18 AM
To: STIC-EIC1600/2900
Subject: Search Request, Case/Application No.: 10/585,892

10585892--S
ictureSearch.p

Requester: NELSON BLAKELY III (P/1614)
Art Unit: GROUP ART UNIT 1614
Employee Number: 84937
Office Location: REM 3B69
Phone Number: (571)270-3290

Case/Application number: 10/585,892
Priority Filing Date:
Format for Search Results: Score
Meaning of unusual acronyms or initialisms:

Identify the novelty:

Additional comments:
Attached you will find the compound structure and chemicle name highlighted.

Attachment: Yes (10585892--StructureSearch.pdf)

RECEIVED
SEP 30 2008
(Initial)

96

LB

Searcher:
Searcher Phone:

Type of Search
NA #: AA#:

Vendors/cost where applicable
STN:

```
=> d his nofile l1-l2; d que stat l2; d his nofile l3-

     (FILE 'REGISTRY' ENTERED AT 09:01:54 ON 02 OCT 2008)
                DEL HIS Y
                ACT NELSON/A
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L1               STR
L2            84 SEA CSS FUL L1


L1               STR
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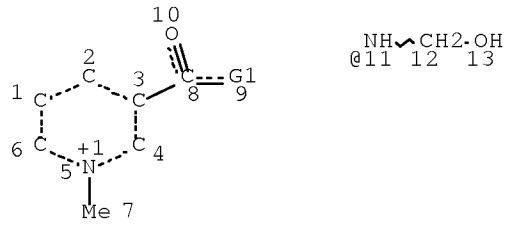


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VAR G1=11/NH2/CH3
NODE ATTRIBUTES:
CHARGE  IS E+1    AT   5
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS  13

STEREO ATTRIBUTES: NONE
L2            84 SEA FILE=REGISTRY CSS FUL L1

100.0% PROCESSED   10333 ITERATIONS                                84 ANSWERS
SEARCH TIME: 00.00.01


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L3            61 SEA ABB=ON  PLU=ON  L2 AND 2=NC
L4            12 SEA ABB=ON  PLU=ON  L2 AND 1=NC
                  D SCAN

     FILE 'CAPLUS' ENTERED AT 09:10:54 ON 02 OCT 2008
L5          1256 SEA ABB=ON  PLU=ON  L2
L6            44 SEA ABB=ON  PLU=ON  L5 (L) (PAC OR THU OR USES)/RL
L7           125 SEA ABB=ON  PLU=ON  L5 AND (63 OR 1)/SC,SX
L8           144 SEA ABB=ON  PLU=ON  L7 OR L6
L9           238 SEA ABB=ON  PLU=ON  GEBICKI J?/AU
L10           81 SEA ABB=ON  PLU=ON  CHLOPICKI S?/AU
L11          313 SEA ABB=ON  PLU=ON  (L9 OR L10)
L12           14 SEA ABB=ON  PLU=ON  L11 AND L5
L13          115 SEA ABB=ON  PLU=ON  L8 AND (PY<2005 OR AY<2005 OR PRY<2005)
L14       712628 SEA ABB=ON  PLU=ON  CARDIO?/OBI OR VASOPROTEC?/OBI OR VASCULAR?
                  /OBI OR HEART/OBI OR ISCHEMI?/OBI OR CORONARY/OBI OR CARDIAC/OB
                  I OR ATHEROSCLER?/OBI OR ANTIARTERIOSCLER?/OBI OR VESSEL?/OBI
                  OR ARTERY/OBI OR OXIDATIVE/OBI (L) STRESS/OBI
L15            2 SEA ABB=ON  PLU=ON  L14 AND L13
```

```
                D SCA TI
L16       203215 SEA ABB=ON  PLU=ON  HYPERTEN?/OBI OR CARDIOVASC?/OBI OR
                 HYPERTRIGLYCER?/OBI OR HYPERCHOLES?/OBI OR  VEIN/OBI OR
                 THROMBOSIS/OBI
L17            1 SEA ABB=ON  PLU=ON  L16 AND L13
L18            2 SEA ABB=ON  PLU=ON  L15 OR L17
L19           13 SEA ABB=ON  PLU=ON  L12 NOT L18
                 D SCAN TI
L20        27519 SEA ABB=ON  PLU=ON  HYPERCHOLESTER?/OBI OR HDL/OBI OR PGI#/OBI
                 OR PROSTACYLIN/OBI
L21            1 SEA ABB=ON  PLU=ON  L13 AND L20
L22            2 SEA ABB=ON  PLU=ON  L21 OR L18
L23           23 SEA ABB=ON  PLU=ON  L5 (L) (THU OR PAC)/RL
L24            5 SEA ABB=ON  PLU=ON  L23 AND (PY<2005 OR AY<2005 OR PRY<2005)
L25            5 SEA ABB=ON  PLU=ON  L24 OR L22
L26           11 SEA ABB=ON  PLU=ON  L12 NOT L25

     FILE 'MEDLINE' ENTERED AT 10:06:48 ON 02 OCT 2008
L27          132 SEA ABB=ON  PLU=ON  L2
                 E NIACINAMIDE/CT
                 E E3+AKK
                 E E3+ALLL
                 E E3+ALL
                 D CT 7
L28         5416 SEA ABB=ON  PLU=ON  NIACINAMIDE/CT
                 E VASOPROTECTIVE/CT
L29           52 SEA ABB=ON  PLU=ON  VASOPROTECT?/TI
L30      2244898 SEA ABB=ON  PLU=ON  L14 OR L16 OR L20
L31           12 SEA ABB=ON  PLU=ON  L30 AND L27
L32           97 SEA ABB=ON  PLU=ON  GEBICKI J?/AU
L33           74 SEA ABB=ON  PLU=ON  CHLOPICKI S?/AU
L34          168 SEA ABB=ON  PLU=ON  (L32 OR L33)
L35            6 SEA ABB=ON  PLU=ON  L34 AND L27
L36            6 SEA ABB=ON  PLU=ON  L34 AND L28
L37            6 SEA ABB=ON  PLU=ON  (L35 OR L36)
L38            3 SEA ABB=ON  PLU=ON  L37 NOT L31

     FILE 'EMBASE' ENTERED AT 10:13:35 ON 02 OCT 2008
L39          210 SEA ABB=ON  PLU=ON  L2
L40          712 SEA ABB=ON  PLU=ON  METHYLNICOTINAMIDE
L41          712 SEA ABB=ON  PLU=ON  L40 OR L39
L42           98 SEA ABB=ON  PLU=ON  GEBICKI J?/AU
L43           72 SEA ABB=ON  PLU=ON  CHLOPICKI S?/AU
L44          168 SEA ABB=ON  PLU=ON  (L42 OR L43)
L45            9 SEA ABB=ON  PLU=ON  L44 AND L41
                 D TRIAL
                 D TRIAL 2-10
L46      2044320 SEA ABB=ON  PLU=ON  L14 OR L16 OR L20
L47           35 SEA ABB=ON  PLU=ON  L46 AND L41
                 D SCAN TI
                 D TRIAL 1-10
L48           22 SEA ABB=ON  PLU=ON  L47 AND PY<2005
                 D TRIAL 1-10
L49            9 SEA ABB=ON  PLU=ON  L45 NOT L48



     FILE 'CAPLUS, MEDLINE, EMBASE' ENTERED AT 10:19:35 ON 02 OCT 2008
L50           32 DUP REM L25 L37 L48 (1 DUPLICATE REMOVED)
                   ANSWERS '1-5' FROM FILE CAPLUS
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                    ANSWERS '6-10' FROM FILE MEDLINE
                    ANSWERS '11-32' FROM FILE EMBASE
L51          14 DUP REM L26 L38 L49 (9 DUPLICATES REMOVED)
                    ANSWERS '1-11' FROM FILE CAPLUS
                    ANSWERS '12-14' FROM FILE MEDLINE
```

```
=> fil reg
FILE 'REGISTRY' ENTERED AT 10:20:27 ON 02 OCT 2008
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provided by InfoChem.

STRUCTURE FILE UPDATES:    1 OCT 2008  HIGHEST RN 1056151-32-6
DICTIONARY FILE UPDATES:   1 OCT 2008  HIGHEST RN 1056151-32-6

New CAS Information Use Policies, enter HELP USAGETERMS for details.

TSCA INFORMATION NOW CURRENT THROUGH July 5, 2008.

  Please note that search-term pricing does apply when
  conducting SmartSELECT searches.

REGISTRY includes numerically searchable data for experimental and
predicted properties as well as tags indicating availability of
experimental property data in the original document.  For information
on property searching in REGISTRY, refer to:

http://www.cas.org/support/stngen/stndoc/properties.html

=> d que stat l2
L1               STR
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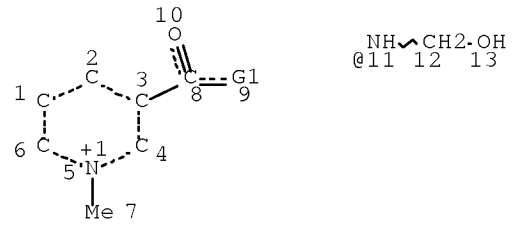


```
VAR G1=11/NH2/CH3
NODE ATTRIBUTES:
CHARGE  IS E+1    AT   5
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS  13

STEREO ATTRIBUTES: NONE
L2           84 SEA FILE=REGISTRY CSS FUL L1

100.0% PROCESSED   10333 ITERATIONS                          84 ANSWERS
SEARCH TIME: 00.00.01



=> fil caplus medline embase
```

```
FILE 'CAPLUS' ENTERED AT 10:20:35 ON 02 OCT 2008
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FILE 'MEDLINE' ENTERED AT 10:20:35 ON 02 OCT 2008

FILE 'EMBASE' ENTERED AT 10:20:35 ON 02 OCT 2008
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=> d que nos l50
L1              STR
L2           84 SEA FILE=REGISTRY CSS FUL L1
L5         1256 SEA FILE=CAPLUS ABB=ON  PLU=ON  L2
L6           44 SEA FILE=CAPLUS ABB=ON  PLU=ON  L5 (L) (PAC OR THU OR USES)/RL

L7          125 SEA FILE=CAPLUS ABB=ON  PLU=ON  L5 AND (63 OR 1)/SC,SX
L8          144 SEA FILE=CAPLUS ABB=ON  PLU=ON  L7 OR L6
L13         115 SEA FILE=CAPLUS ABB=ON  PLU=ON  L8 AND (PY<2005 OR AY<2005 OR
                PRY<2005)
L14      712628 SEA FILE=CAPLUS ABB=ON  PLU=ON  CARDIO?/OBI OR VASOPROTEC?/OBI
                OR VASCULAR?/OBI OR HEART/OBI OR ISCHEMI?/OBI OR CORONARY/OBI
                OR CARDIAC/OBI OR ATHEROSCLER?/OBI OR ANTIARTERIOSCLER?/OBI OR
                VESSEL?/OBI OR ARTERY/OBI OR OXIDATIVE/OBI (L) STRESS/OBI
L15           2 SEA FILE=CAPLUS ABB=ON  PLU=ON  L14 AND L13
L16      203215 SEA FILE=CAPLUS ABB=ON  PLU=ON  HYPERTEN?/OBI OR CARDIOVASC?/OB
                I OR HYPERTRIGLYCER?/OBI OR HYPERCHOLES?/OBI OR  VEIN/OBI OR
                THROMBOSIS/OBI
L17           1 SEA FILE=CAPLUS ABB=ON  PLU=ON  L16 AND L13
L18           2 SEA FILE=CAPLUS ABB=ON  PLU=ON  L15 OR L17
L20       27519 SEA FILE=CAPLUS ABB=ON  PLU=ON  HYPERCHOLESTER?/OBI OR HDL/OBI
                OR PGI#/OBI OR PROSTACYLIN/OBI
L21           1 SEA FILE=CAPLUS ABB=ON  PLU=ON  L13 AND L20
L22           2 SEA FILE=CAPLUS ABB=ON  PLU=ON  L21 OR L18
L23          23 SEA FILE=CAPLUS ABB=ON  PLU=ON  L5 (L) (THU OR PAC)/RL
L24           5 SEA FILE=CAPLUS ABB=ON  PLU=ON  L23 AND (PY<2005 OR AY<2005 OR
                PRY<2005)
L25           5 SEA FILE=CAPLUS ABB=ON  PLU=ON  L24 OR L22
L27         132 SEA FILE=MEDLINE ABB=ON  PLU=ON  L2
L28        5416 SEA FILE=MEDLINE ABB=ON  PLU=ON  NIACINAMIDE/CT
L32          97 SEA FILE=MEDLINE ABB=ON  PLU=ON  GEBICKI J?/AU
L33          74 SEA FILE=MEDLINE ABB=ON  PLU=ON  CHLOPICKI S?/AU
L34         168 SEA FILE=MEDLINE ABB=ON  PLU=ON  (L32 OR L33)
L35           6 SEA FILE=MEDLINE ABB=ON  PLU=ON  L34 AND L27
L36           6 SEA FILE=MEDLINE ABB=ON  PLU=ON  L34 AND L28
L37           6 SEA FILE=MEDLINE ABB=ON  PLU=ON  (L35 OR L36)
L39         210 SEA FILE=EMBASE ABB=ON  PLU=ON  L2
L40         712 SEA FILE=EMBASE ABB=ON  PLU=ON  METHYLNICOTINAMIDE
L41         712 SEA FILE=EMBASE ABB=ON  PLU=ON  L40 OR L39
L46     2044320 SEA FILE=EMBASE ABB=ON  PLU=ON  L14 OR L16 OR L20
L47          35 SEA FILE=EMBASE ABB=ON  PLU=ON  L46 AND L41
L48          22 SEA FILE=EMBASE ABB=ON  PLU=ON  L47 AND PY<2005
L50          32 DUP REM L25 L37 L48 (1 DUPLICATE REMOVED)


=> d que nos l51
L1              STR
L2           84 SEA FILE=REGISTRY CSS FUL L1
L5         1256 SEA FILE=CAPLUS ABB=ON  PLU=ON  L2
L6           44 SEA FILE=CAPLUS ABB=ON  PLU=ON  L5 (L) (PAC OR THU OR USES)/RL
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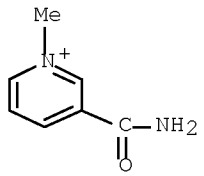
L7          125 SEA FILE=CAPLUS ABB=ON  PLU=ON  L5 AND (63 OR 1)/SC,SX
L8          144 SEA FILE=CAPLUS ABB=ON  PLU=ON  L7 OR L6
L9          238 SEA FILE=CAPLUS ABB=ON  PLU=ON  GEBICKI J?/AU
L10          81 SEA FILE=CAPLUS ABB=ON  PLU=ON  CHLOPICKI S?/AU
L11         313 SEA FILE=CAPLUS ABB=ON  PLU=ON  (L9 OR L10)
L12          14 SEA FILE=CAPLUS ABB=ON  PLU=ON  L11 AND L5
L13         115 SEA FILE=CAPLUS ABB=ON  PLU=ON  L8 AND (PY<2005 OR AY<2005 OR
                PRY<2005)
L14      712628 SEA FILE=CAPLUS ABB=ON  PLU=ON  CARDIO?/OBI OR VASOPROTEC?/OBI
                OR VASCULAR?/OBI OR HEART/OBI OR ISCHEMI?/OBI OR CORONARY/OBI
                OR CARDIAC/OBI OR ATHEROSCLER?/OBI OR ANTIARTERIOSCLER?/OBI OR
                VESSEL?/OBI OR ARTERY/OBI OR OXIDATIVE/OBI (L) STRESS/OBI
L15           2 SEA FILE=CAPLUS ABB=ON  PLU=ON  L14 AND L13
L16      203215 SEA FILE=CAPLUS ABB=ON  PLU=ON  HYPERTEN?/OBI OR CARDIOVASC?/OB
                I OR HYPERTRIGLYCER?/OBI OR HYPERCHOLES?/OBI OR  VEIN/OBI OR
                THROMBOSIS/OBI
L17           1 SEA FILE=CAPLUS ABB=ON  PLU=ON  L16 AND L13
L18           2 SEA FILE=CAPLUS ABB=ON  PLU=ON  L15 OR L17
L20       27519 SEA FILE=CAPLUS ABB=ON  PLU=ON  HYPERCHOLESTER?/OBI OR HDL/OBI
                OR PGI#/OBI OR PROSTACYLIN/OBI
L21           1 SEA FILE=CAPLUS ABB=ON  PLU=ON  L13 AND L20
L22           2 SEA FILE=CAPLUS ABB=ON  PLU=ON  L21 OR L18
L23          23 SEA FILE=CAPLUS ABB=ON  PLU=ON  L5 (L) (THU OR PAC)/RL
L24           5 SEA FILE=CAPLUS ABB=ON  PLU=ON  L23 AND (PY<2005 OR AY<2005 OR
                PRY<2005)
L25           5 SEA FILE=CAPLUS ABB=ON  PLU=ON  L24 OR L22
L26          11 SEA FILE=CAPLUS ABB=ON  PLU=ON  L12 NOT L25
L27         132 SEA FILE=MEDLINE ABB=ON  PLU=ON  L2
L28        5416 SEA FILE=MEDLINE ABB=ON  PLU=ON  NIACINAMIDE/CT
L30     2244898 SEA FILE=MEDLINE ABB=ON  PLU=ON  L14 OR L16 OR L20
L31          12 SEA FILE=MEDLINE ABB=ON  PLU=ON  L30 AND L27
L32          97 SEA FILE=MEDLINE ABB=ON  PLU=ON  GEBICKI J?/AU
L33          74 SEA FILE=MEDLINE ABB=ON  PLU=ON  CHLOPICKI S?/AU
L34         168 SEA FILE=MEDLINE ABB=ON  PLU=ON  (L32 OR L33)
L35           6 SEA FILE=MEDLINE ABB=ON  PLU=ON  L34 AND L27
L36           6 SEA FILE=MEDLINE ABB=ON  PLU=ON  L34 AND L28
L37           6 SEA FILE=MEDLINE ABB=ON  PLU=ON  (L35 OR L36)
L38           3 SEA FILE=MEDLINE ABB=ON  PLU=ON  L37 NOT L31
L39         210 SEA FILE=EMBASE ABB=ON  PLU=ON  L2
L40         712 SEA FILE=EMBASE ABB=ON  PLU=ON  METHYLNICOTINAMIDE
L41         712 SEA FILE=EMBASE ABB=ON  PLU=ON  L40 OR L39
L42          98 SEA FILE=EMBASE ABB=ON  PLU=ON  GEBICKI J?/AU
L43          72 SEA FILE=EMBASE ABB=ON  PLU=ON  CHLOPICKI S?/AU
L44         168 SEA FILE=EMBASE ABB=ON  PLU=ON  (L42 OR L43)
L45           9 SEA FILE=EMBASE ABB=ON  PLU=ON  L44 AND L41
L46     2044320 SEA FILE=EMBASE ABB=ON  PLU=ON  L14 OR L16 OR L20
L47          35 SEA FILE=EMBASE ABB=ON  PLU=ON  L46 AND L41
L48          22 SEA FILE=EMBASE ABB=ON  PLU=ON  L47 AND PY<2005
L49           9 SEA FILE=EMBASE ABB=ON  PLU=ON  L45 NOT L48
L51          14 DUP REM L26 L38 L49 (9 DUPLICATES REMOVED)


=> d .ca hitstr l50 1-5; d ibib ab ct l50 6-32; d ibib l51 1-14

L50  ANSWER 1 OF 32  CAPLUS  COPYRIGHT 2008 ACS on STN DUPLICATE 1
ACCESSION NUMBER:         2003:643918  CAPLUS  Full-text
DOCUMENT NUMBER:          140:70586
TITLE:                    1-Methylnicotinamide: a potent anti-inflammatory agent
                          of vitamin origin
```

AUTHOR(S): Gebicki, Jerzy; Sysa-Jedrzejowska, Anna; Adamus, Jan; Wozniacka, Anna; Rybak, Malgorzata; Zielonka, Jacek
CORPORATE SOURCE: Institute of Applied Radiation Chemistry, Technical University, Lodz, PL 90-924, Pol.
SOURCE: Polish Journal of Pharmacology (2003), 55(1), 109-112
CODEN: PJPAE3; ISSN: 1230-6002
PUBLISHER: Polish Academy of Sciences, Institute of Pharmacology
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 19 Aug 2003
AB It has been found that 1-methylnicotinamide (MNA+), a metabolite of nicotinamide, possesses significant anti-inflammatory properties. MNA+ is chemical stable, non-toxic and well tolerated. MNA+ can be used to treat wide variety of diseases and disorders and the use of this compound provides certain advantages over the use of nicotinamide.
CC 1-7 (Pharmacology)
IT 3106-60-3, 1-Methylnicotinamide
RL: DMA (Drug mechanism of action); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(1-methylnicotinamide anti-inflammatory effects in human skin disorders and possible mechanisms)
IT 3106-60-3, 1-Methylnicotinamide
RL: DMA (Drug mechanism of action); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(1-methylnicotinamide anti-inflammatory effects in human skin disorders and possible mechanisms)
RN 3106-60-3 CAPLUS
CN Pyridinium, 3-(aminocarbonyl)-1-methyl- (CA INDEX NAME)



REFERENCE COUNT: 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L50 ANSWER 2 OF 32 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2006:211502 CAPLUS Full-text
DOCUMENT NUMBER: 144:267270
TITLE: Fused bicyclic natural compounds and their use as inhibitors of PARP and PARP-mediated inflammatory processes
INVENTOR(S): Hageman, Gerrigje Johanna; Moonen, Harald Johan Joseph; Geraets, Liesbeth; Bast, Aalt; Wouters, Emiel Frans Maria
PATENT ASSIGNEE(S): Stichting voor de Technische Wetenschappen, Neth.
SOURCE: PCT Int. Appl., 93 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006024545	A1	20060309	WO 2005-EP9514	20050905 <--

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRIORITY APPLN. INFO.: EP 2004-447196 A 20040903 <--
EP 2004-447238 A 20041028 <--

OTHER SOURCE(S): MARPAT 144:267270

ED Entered STN: 09 Mar 2006

AB The invention relates to the use of at least two compds., of which the first compound is a natural compound such as xanthines, coumarins, flavonoids, and anthocyanidins which are identified as PARP-1 (poly(ADP-ribose) polymerase 1) inhibitors and a second compound, which is an NAD+ precursor for preparing medicaments, medical foods or nutraceuticals. The invention also relates to the use of these compds. or pharmaceutical compns. comprising at least two of these compds. as anti-inflammatory agent for treating acute or chronic inflammation in certain diseases or disorders.

CC 1-7 (Pharmacology)

IT Antiarteriosclerotics
(antiatherosclerotics; fused bicyclic natural compds. and their use as inhibitors of PARP and PARP-mediated inflammatory processes and combination with NAD+ precursors)

IT Anti-inflammatory agents
Anti-ischemic agents
Antidiabetic agents
Antifibrotic agents
Antirheumatic agents
Antitumor agents
Atherosclerosis
Autoimmune disease
Combination chemotherapy
Diabetes mellitus
Dietary supplements
Fibrosis
Human
Inflammation
Ischemia
Neoplasm
Rheumatoid arthritis
(fused bicyclic natural compds. and their use as inhibitors of PARP and PARP-mediated inflammatory processes and combination with NAD+ precursors)

IT 50-89-5, Thymidine, biological studies 50-89-5D, Thymidine, derivs. and esters metabolites and prodrugs 58-08-2, Caffeine, biological studies 58-08-2D, Caffeine, derivs. and esters metabolites and prodrugs 58-55-9, Theophylline, biological studies 58-55-9D, Theophylline, derivs. and esters metabolites and prodrugs 59-67-6, Nicotinic acid, biological studies 59-67-6D, Nicotinic acid, derivs. and esters metabolites and prodrugs 68-94-0, Hypoxanthine 73-22-3, L-Tryptophan, biological

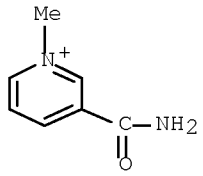
studies 73-22-3D, L-Tryptophan, derivs. and esters metabolites and prodrugs 81-54-9, Purpurin 81-54-9D, Purpurin, derivs. and esters metabolites and prodrugs 82-02-0, Khellin 82-02-0D, Khellin, derivs. and esters metabolites and prodrugs 83-67-0, Theobromine 83-67-0D, Theobromine, derivs. and esters metabolites and prodrugs 91-64-5, Coumarin 91-64-5D, Coumarin, derivs. and esters metabolites and prodrugs 93-35-6, Umbelliferone 93-35-6D, Umbelliferone, derivs. and esters metabolites and prodrugs 97-59-6, Allantoin 97-59-6D, Allantoin, derivs. and esters metabolites and prodrugs 98-92-0, Nicotinamide 98-92-0D, Nicotinamide, derivs. and esters metabolites and prodrugs 117-39-5, Quercetin 117-39-5D, Quercetin, derivs. and esters metabolites and prodrugs 120-08-1, Scoparone 120-08-1D, Scoparone, derivs. and esters metabolites and prodrugs 134-01-0, Peonidin 134-01-0D, Peonidin, derivs. and esters metabolites and prodrugs 134-04-3, Pelargonidin 134-04-3D, Pelargonidin, derivs. and esters metabolites and prodrugs 140-10-3, trans-Cinnamic acid, biological studies 140-10-3D, trans-Cinnamic acid, derivs. and esters metabolites and prodrugs 154-23-4, Catechin 154-23-4D, Catechin, derivs. and esters metabolites and prodrugs 218-01-9, Chrysene 298-81-7, 8-Methoxypsoralen 298-81-7D, 8-Methoxypsoralen, derivs. and esters metabolites and prodrugs 305-01-1, Esculetin 305-01-1D, Esculetin, derivs. and esters metabolites and prodrugs 305-84-0, Carnosine 315-30-0, Allopurinol 327-97-9, Chlorogenic acid 327-97-9D, Chlorogenic acid, derivs. and esters metabolites and prodrugs 331-39-5, Caffeic acid 331-39-5D, Caffeic acid, derivs. and esters metabolites and prodrugs 446-72-0, Genistein 458-37-7, Curcumin 471-53-4, 18β-Glycyrrhetinic acid 471-53-4D, 18β-Glycyrrhetinic acid, derivs. and esters metabolites and prodrugs 473-98-3, Betulin 473-98-3D, Betulin, derivs. and esters metabolites and prodrugs 476-66-4, Ellagic acid 476-66-4D, Ellagic acid, derivs. and esters metabolites and prodrugs 479-13-0, Coumestrol 479-13-0D, Coumestrol, derivs. and esters metabolites and prodrugs 480-16-0, Morin 480-16-0D, Morin, derivs. and esters metabolites and prodrugs 480-18-2, Taxifolin 480-18-2D, Taxifolin, derivs. and esters metabolites and prodrugs 480-41-1, Naringenin 480-41-1D, Naringenin, derivs. and esters metabolites and prodrugs 486-35-1, Daphnetin 486-35-1D, Daphnetin, derivs. and esters metabolites and prodrugs 486-66-8, Daidzein 486-66-8D, Daidzein, derivs. and esters metabolites and prodrugs 487-36-5, Pinoresinol 489-35-0, Gossypetin 489-35-0D, Gossypetin, derivs. and esters metabolites and prodrugs 490-46-0, (-)-Epicatechin 490-46-0D, (-)-Epicatechin, derivs. and esters metabolites and prodrugs 490-91-5, Thymoquinone 490-91-5D, Thymoquinone, derivs. and esters metabolites and prodrugs 491-67-8, Baicalein 491-67-8D, Baicalein, derivs. and esters metabolites and prodrugs 491-70-3, Luteolin 495-02-3 495-02-3D, derivs. and esters metabolites and prodrugs 501-36-0, Resveratrol 518-28-5, Podophyllotoxin 518-29-6, β-Peltatin 518-82-1, Emodin 518-82-1D, Emodin, derivs. and esters metabolites and prodrugs 520-18-3, Kaempferol 520-18-3D, Kaempferol, derivs. and esters metabolites and prodrugs 520-31-0, Tricetin 520-31-0D, Tricetin, derivs. and esters metabolites and prodrugs 520-36-5, Apigenin 522-12-3, Quercitrin 522-12-3D, Quercitrin, derivs. and esters metabolites and prodrugs 525-82-6, Flavone 525-82-6D, Flavone, derivs. and esters metabolites and prodrugs 528-48-3, Fisetin 528-48-3D, Fisetin, derivs. and esters metabolites and prodrugs 528-53-0, Delphinidin 528-53-0D, Delphinidin, derivs. and esters metabolites and prodrugs 528-58-5, Cyanidin 528-58-5D, Cyanidin, derivs. and esters metabolites and prodrugs 529-44-2, Cannabiscetin 529-44-2D, Cannabiscetin, derivs. and esters metabolites and prodrugs 529-84-0, 6,7-Dihydroxy-4-methylcoumarin 529-84-0D, 6,7-Dihydroxy-4-methylcoumarin, derivs. and esters metabolites

and prodrugs 531-81-7, Coumarin-3-carboxylic acid 531-81-7D, Coumarin-3-carboxylic acid, derivs. and esters metabolites and prodrugs 535-83-1D, Trigonelline, derivs. and metabolites and pyrolysis products 545-46-0, Uvaol 545-46-0D, Uvaol, derivs. and esters metabolites and prodrugs 545-47-1, Lupeol 545-47-1D, Lupeol, derivs. and esters metabolites and prodrugs 548-83-4, Galangin 548-83-4D, Galangin, derivs. and esters metabolites and prodrugs 574-84-5, Fraxetin 574-84-5D, Fraxetin, derivs. and esters metabolites and prodrugs 580-72-3, Matairesinol 583-17-5, o-Coumaric acid 583-17-5D, o-Coumaric acid, derivs. and esters metabolites and prodrugs 588-30-7, m-Coumaric acid 588-30-7D, m-Coumaric acid, derivs. and esters metabolites and prodrugs 611-59-6, Paraxanthine 611-59-6D, Paraxanthine, derivs. and esters metabolites and prodrugs 643-84-5, Malvidin 643-84-5D, Malvidin, derivs. and esters metabolites and prodrugs 694-56-4, 1-Methylpyridinium 694-56-4D, 1-Methylpyridinium, derivs. and esters metabolites and prodrugs 701-44-0 701-44-0D, derivs. and prodrugs 708-79-2, 1-Methyluric acid 708-79-2D, 1-Methyluric acid, derivs. and esters metabolites and prodrugs 769-49-3, 1-Methyl-4-pyridone-5-carboxamide 769-49-3D, 1-Methyl-4-pyridone-5-carboxamide, derivs. and esters metabolites and prodrugs 779-30-6, 3-Acetamidocoumarin 779-30-6D, 3-Acetamidocoumarin, derivs. and esters metabolites and prodrugs 833-68-1, 6-Acetamidocoumarin 833-68-1D, 6-Acetamidocoumarin, derivs. and esters metabolites and prodrugs 961-29-5, Isoliquiritigenin 961-29-5D, Isoliquiritigenin, derivs. and esters metabolites and prodrugs 989-51-5, (-)-Epigallocatechin gallate 989-51-5D, (-)-Epigallocatechin gallate, derivs. and esters metabolites and prodrugs 1063-77-0, Nomilin 1063-77-0D, Nomilin, derivs. and esters metabolites and prodrugs 1076-38-6, 4-Hydroxycoumarin 1076-38-6D, 4-Hydroxycoumarin, derivs. and esters metabolites and prodrugs 1429-30-7, Petunidin 1429-30-7D, Petunidin, derivs. and esters metabolites and prodrugs 1449-05-4, 18α-Glycyrrhetinic acid 1449-05-4D, 18α-Glycyrrhetinic acid, derivs. and esters metabolites and prodrugs 1453-82-3, Isonicotinamide 1617-53-4, Amentoflavone 1617-53-4D, Amentoflavone, derivs. and esters metabolites and prodrugs 1617-72-7, Allobetulin 1617-72-7D, Allobetulin, derivs. and esters metabolites and prodrugs 2107-76-8, 5,7-Dihydroxy-4-methylcoumarin 2107-76-8D, 5,7-Dihydroxy-4-methylcoumarin, derivs. and esters metabolites and prodrugs 2107-77-9, 4-Methyldaphnetin 2107-77-9D, 4-Methyldaphnetin, derivs. and esters metabolites and prodrugs 2465-59-0, Oxypurinol 2465-59-0D, Oxypurinol, derivs. and esters metabolites and prodrugs 3106-60-3, 1-Methylnicotinamide 3106-60-3D, 1-Methylnicotinamide, derivs. and esters metabolites and prodrugs 3544-24-9, 3-Aminobenzamide 3650-73-5, L-Homocarnosine 4707-32-8, β-Lapachone 4707-32-8D, β-Lapachone, derivs. and esters metabolites and prodrugs 6136-37-4, 1-Methylxanthine 6136-37-4D, 1-Methylxanthine, derivs. and esters metabolites and prodrugs 6805-41-0, Aescin 7400-08-0, p-Coumaric acid 10236-47-2, Naringin 10236-47-2D, Naringin, derivs. and esters metabolites and prodrugs 16969-45-2D, Pyridinium, alkyl derivs. 18241-35-5, 1,4-Dimethylpyridinium 18241-35-5D, 1,4-Dimethylpyridinium, derivs. and esters metabolites and prodrugs 19186-35-7, Deoxypodophyllotoxin 27003-73-2, Lariciresinol 29388-59-8, Secoisolariciresinol 33868-03-0, 1,7-Dimethyluric acid 33868-03-0D, 1,7-Dimethyluric acid, derivs. and esters metabolites and prodrugs 40456-50-6, Yatein 73465-37-9 78473-71-9, Enterolactone 80226-00-2, Enterodiol 128443-52-7 347359-71-1 878045-11-5 878045-11-5D, derivs. and prodrugs 878045-12-6 878045-13-7 878045-14-8 878045-15-9 878045-16-0 878045-17-1 878045-18-2 878045-19-3 878045-20-6 878045-21-7 878045-22-8 878045-23-9 878045-24-0 878045-25-1 878045-26-2 878045-27-3

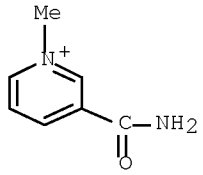
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(fused bicyclic natural compds. and their use as inhibitors of PARP and PARP-mediated inflammatory processes and combination with NAD+ precursors)

IT 3106-60-3, 1-Methylnicotinamide 3106-60-3D, 1-Methylnicotinamide, derivs. and esters metabolites and prodrugs
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(fused bicyclic natural compds. and their use as inhibitors of PARP and PARP-mediated inflammatory processes and combination with NAD+ precursors)

RN 3106-60-3 CAPLUS
CN Pyridinium, 3-(aminocarbonyl)-1-methyl- (CA INDEX NAME)



RN 3106-60-3 CAPLUS
CN Pyridinium, 3-(aminocarbonyl)-1-methyl- (CA INDEX NAME)



REFERENCE COUNT: 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

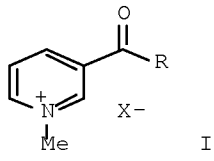
L50 ANSWER 3 OF 32 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2005:673111 CAPLUS Full-text
DOCUMENT NUMBER: 143:146685
TITLE: The use of quaternary pyridinium salts as vasoprotective agents
INVENTOR(S): Gebicki, Jerzy; Chlopicki, Stefan
PATENT ASSIGNEE(S): Pharmena Sp Z O.O., Pol.
SOURCE: PCT Int. Appl., 30 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005067927	A2	20050728	WO 2005-EP50057	20050107 <--
WO 2005067927	A3	20051201		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, SM
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2005205066	A1	20050728	AU 2005-205066		20050107 <--
CA 2547234	A1	20050728	CA 2005-2547234		20050107 <--
EP 1713480	A2	20061025	EP 2005-726215		20050107 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK, IS					
CN 1905875	A	20070131	CN 2005-80001712		20050107 <--
JP 2007517833	T	20070705	JP 2006-548308		20050107 <--
MX 2006PA07930	A	20060926	MX 2006-PA7930		20060711 <--
US 20080207702	A1	20080828	US 2006-585892		20060711 <--
PRIORITY APPLN. INFO.:			PL 2004-364348	A	20040112 <--
			WO 2005-EP50057	W	20050107

OTHER SOURCE(S): MARPAT 143:146685
ED Entered STN: 29 Jul 2005
GI



I

AB The invention relates to the use of quaternary pyridinium salts I [R = NH2, CH3, N(H)CH2OH; X = pharmaceutically acceptable counterion] for the preparation of vasoprotective agents for the treatment or prevention of conditions or diseases associated with dysfunction of vascular endothelium, oxidative stress, and/or insufficient production of endothelial prostacyclin PGI2, in particular but not exclusively if the above coincides with hypercholesterolemia, hypertriglyceridemia or low HDL level.

IC ICM A61K031-4425
ICS A61P001-04; A61P001-16; A61P003-04; A61P003-10; A61P009-00; A61P009-10; A61P009-12; A61P011-00; A61P015-10; A61P025-16; A61P025-28; A61P029-00; A61P031-00; A61P041-00

CC 1-8 (Pharmacology)

ST quaternary pyridinium salt vasoprotective agent; vascular endothelium dysfunction quaternary pyridinium salt; oxidative stress quaternary pyridinium salt; PGI2 prodn insufficiency quaternary pyridinium salt; hypercholesterolemia hypertriglyceridemia low HDL level quaternary pyridinium salt

IT Prostaglandins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(I; quaternary pyridinium salts for vasoprotective agents)

IT Neutrophil
(activation; quaternary pyridinium salts for vasoprotective agents)

IT Respiratory distress syndrome
(adult; quaternary pyridinium salts for vasoprotective agents)
IT Blood vessel, disease
(allograft vasculopathy; quaternary pyridinium salts for vasoprotective agents)
IT Transplant and Transplantation
(allotransplant, vasculopathy; quaternary pyridinium salts for vasoprotective agents)
IT Antiarteriosclerotics
(antiatherosclerotics; quaternary pyridinium salts for vasoprotective agents)
IT Brain, disease
(cerebrovascular, ischemia; quaternary pyridinium salts for vasoprotective agents)
IT Heart, disease
(chronic coronary disease; quaternary pyridinium salts for vasoprotective agents)
IT Lung, disease
(chronic obstructive pulmonary disease; quaternary pyridinium salts for vasoprotective agents)
IT Liver, disease
(chronic; quaternary pyridinium salts for vasoprotective agents)
IT Artery, disease
(coronary, unstable; quaternary pyridinium salts for vasoprotective agents)
IT Nervous system, disease
(degeneration; quaternary pyridinium salts for vasoprotective agents)
IT Mental and behavioral disorders
(dementia, vascular; quaternary pyridinium salts for vasoprotective agents)
IT Blood vessel, disease
(diabetic microangiopathy; quaternary pyridinium salts for vasoprotective agents)
IT Kidney, disease
(diabetic nephropathy; quaternary pyridinium salts for vasoprotective agents)
IT Nerve, disease
(diabetic neuropathy; quaternary pyridinium salts for vasoprotective agents)
IT Eye, disease
(diabetic retinopathy; quaternary pyridinium salts for vasoprotective agents)
IT Ulcer
(duodenal; quaternary pyridinium salts for vasoprotective agents)
IT Intestine, disease
(duodenum, ulcer; quaternary pyridinium salts for vasoprotective agents)
IT Blood vessel, disease
(endothelium; quaternary pyridinium salts for vasoprotective agents)
IT Circulation
(extracorporeal, surgery with; quaternary pyridinium salts for vasoprotective agents)
IT Heart, disease
Kidney, disease
(failure, chronic; quaternary pyridinium salts for

vasoprotective agents)
IT Ulcer
(gastric; quaternary pyridinium salts for vasoprotective agents)
IT Dialysis
(hemodialysis; quaternary pyridinium salts for vasoprotective agents)
IT Sexual disorders
(impotence; quaternary pyridinium salts for vasoprotective agents)
IT Heart, disease
(infarction; quaternary pyridinium salts for vasoprotective agents)
IT Intestine, disease
(inflammatory; quaternary pyridinium salts for vasoprotective agents)
IT Drug delivery systems
(inhalants; quaternary pyridinium salts for vasoprotective agents)
IT High-density lipoproteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(low level of; quaternary pyridinium salts for vasoprotective agents)
IT Mental and behavioral disorders
Stress, animal
(mental stress; quaternary pyridinium salts for vasoprotective agents)
IT Kidney, disease
(nephrotic syndrome; quaternary pyridinium salts for vasoprotective agents)
IT Cell activation
(neutrophil; quaternary pyridinium salts for vasoprotective agents)
IT Drug tolerance
(nitrate tolerance; quaternary pyridinium salts for vasoprotective agents)
IT Nitrates, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(nitrate tolerance; quaternary pyridinium salts for vasoprotective agents)
IT Drug delivery systems
(oral; quaternary pyridinium salts for vasoprotective agents)
IT Drug delivery systems
(parenterals; quaternary pyridinium salts for vasoprotective agents)
IT Circulation
(peripheral circulation revascularization; quaternary pyridinium salts for vasoprotective agents)
IT Ovary, disease
(polycystic; quaternary pyridinium salts for vasoprotective agents)
IT Amyloidosis
(primary; quaternary pyridinium salts for vasoprotective agents)
IT Hypertension
(pulmonary; quaternary pyridinium salts for vasoprotective agents)
IT Aging, animal
Alzheimer's disease
Anti-Alzheimer's agents

Anti-infective agents
Anti-inflammatory agents
Anti-ischemic agents
Anticholesteremic agents
Anticoagulants
Antidiabetic agents
Antiglaucoma agents
Antihypertensives
Antiobesity agents
Antiparkinsonian agents
Antirheumatic agents
Antiulcer agents
Atherosclerosis
Blood vessel
Cardiovascular agents
Cardiovascular system, disease
Combination chemotherapy
Coronary angioplasty
Coronary bypass surgery
Cystic fibrosis
Diabetes mellitus
Dietary supplements
Gastrointestinal agents
Glaucoma (disease)
Human
Hypercholesterolemia
Hypertension
Hypertriglyceridemia
Hypolipemic agents
Infection
Inflammation
Menopause
Nervous system agents
Obesity
Oxidative stress, biological
Parkinson's disease
Periodontium, disease
Platelet aggregation
Platelet aggregation
Preeclampsia
Prophylaxis
Rheumatoid arthritis
Sickle cell anemia
Sleep apnea
Thrombosis
Tobacco smoke
(quaternary pyridinium salts for vasoprotective agents)

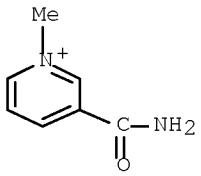
IT Dyslipidemia
Glycerides, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(quaternary pyridinium salts for vasoprotective agents)

IT Behavior
(smoking; quaternary pyridinium salts for vasoprotective agents)

IT Medical goods
(stents; quaternary pyridinium salts for vasoprotective agents)

IT Brain, disease
(stroke; quaternary pyridinium salts for vasoprotective agents)

IT Heart, disease
(sudden cardiac death; quaternary pyridinium salts for vasoprotective agents)
IT Lupus erythematosus
(systemic; quaternary pyridinium salts for vasoprotective agents)
IT Thrombosis
(thromboangiitis obliterans; quaternary pyridinium salts for vasoprotective agents)
IT Embolism
(thromboembolism, venous; quaternary pyridinium salts for vasoprotective agents)
IT Stomach, disease
(ulcer; quaternary pyridinium salts for vasoprotective agents)
IT Endothelium
(vascular, disease; quaternary pyridinium salts for vasoprotective agents)
IT Vein, disease
(venous thromboembolic disease; quaternary pyridinium salts for vasoprotective agents)
IT Infection
(viral hepatitis; quaternary pyridinium salts for vasoprotective agents)
IT Hepatitis
(viral; quaternary pyridinium salts for vasoprotective agents)
IT 6027-13-0, Homocysteine
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(hyperhomocysteinemia; quaternary pyridinium salts for vasoprotective agents)
IT 57-88-5, Cholesterol, biological studies 35121-78-9, PGI2
54397-85-2, TXB2 58962-34-8
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(quaternary pyridinium salts for vasoprotective agents)
IT 59-67-6, Nicotinic acid, biological studies 98-92-0, Nicotinamide
535-83-1, Trigonelline 701-44-0
RL: PAC (Pharmacological activity); BIOL (Biological study)
(quaternary pyridinium salts for vasoprotective agents)
IT 3106-60-3D, 1-Methylnicotinamide, salts 51061-43-9D,
salts 282521-18-0D, salts
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(quaternary pyridinium salts for vasoprotective agents)
IT 9004-10-8, Insulin, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(resistance; quaternary pyridinium salts for vasoprotective agents)
IT 3106-60-3D, 1-Methylnicotinamide, salts 51061-43-9D,
salts 282521-18-0D, salts
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(quaternary pyridinium salts for vasoprotective agents)
RN 3106-60-3 CAPLUS
CN Pyridinium, 3-(aminocarbonyl)-1-methyl- (CA INDEX NAME)



RN 51061-43-9 CAPLUS
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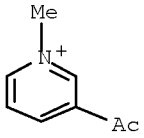

RN 282521-18-0 CAPLUS
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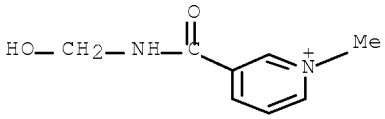
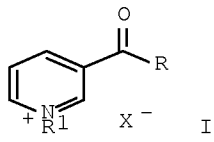


L50 ANSWER 4 OF 32 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2000:475642 CAPLUS Full-text
DOCUMENT NUMBER: 133:109950
TITLE: Pyridine derivatives for the treatment of skin diseases
INVENTOR(S): Gebicki, Jerzy; Sysa-Jedrzejowska, Anna; Adamus, Jan
PATENT ASSIGNEE(S): Technical University of Lodz, Pol.
SOURCE: PCT Int. Appl., 21 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000040559	A1	20000713	WO 2000-IB19	20000107 <--
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
PL 190755	B1	20060131	PL 1999-330768	19990107 <--

EP 1147086	A1	20011024	EP 2000-900030		20000107 <--
EP 1147086	B1	20050330			
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO					
HU 2001004963	A2	20020429	HU 2001-4963		20000107 <--
HU 2001004963	A3	20021128			
HU 225475	B1	20061228			
RU 2199319	C1	20030227	RU 2001-121650		20000107 <--
AT 292117	T	20050415	AT 2000-900030		20000107 <--
ES 2238985	T3	20050916	ES 2000-900030		20000107 <--
SK 285869	B6	20071004	SK 2001-951		20000107 <--
PRIORITY APPLN. INFO.:			PL 1999-330768	A	19990107 <--
			WO 2000-IB19	W	20000107 <--

OTHER SOURCE(S): MARPAT 133:109950
ED Entered STN: 14 Jul 2000
GI



I

AB Disclosed is the use of a compound of formula (I: R represents the group NR2R3 or the group OR4; R1 represents C1-4 alkyl; R2 and R4 each independently represent hydrogen or C1-4 alkyl; R3 represents hydrogen, C1-4 alkyl or CH2OH; and X- is a physiol. suitable counter-anion) in the treatment of skin diseases or disorders, hair loss, sunburn, burns, scalds and for wound healing. Also disclosed are pharmaceutical formulations of compds. of formula I, particularly for topical use.

IC ICM C07D213-80
ICS C07D213-82; A61K031-4406; A61K031-4425; A61K031-455; A61P017-02; A61P017-14

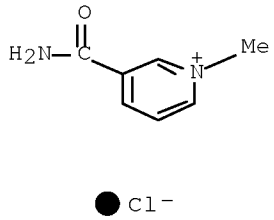
CC 63-6 (Pharmaceuticals)
Section cross-reference(s): 1

IT 50-21-5D, Lactic acid, salts 64-19-7D, Acetic acid, salts, biological studies 65-85-0D, Benzoic acid, salts, biological studies 69-72-7D, Salicylic acid, salts 77-92-9D, Citric acid, salts 1005-24-9, 1-Methylnicotinamide chloride 3106-60-3D, 1-Methylnicotinamide, salts 6138-41-6 46058-12-2 56338-90-0 282521-18-0D, salts 282521-19-1 282521-20-4, biological studies 282521-21-5 , biological studies 282521-22-6 282521-23-7
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PEP (Physical, engineering or chemical process); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(pyridine derivs. for the treatment of skin diseases)

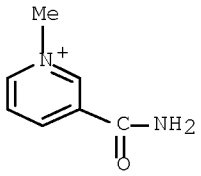
IT 1005-24-9, 1-Methylnicotinamide chloride 3106-60-3D, 1-Methylnicotinamide, salts 282521-18-0D, salts 282521-20-4, biological studies 282521-21-5, biological studies 282521-22-6
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PEP (Physical, engineering or chemical process); THU (Therapeutic use); BIOL (Biological study); PROC (Process);

USES (Uses)
(pyridine derivs. for the treatment of skin diseases)

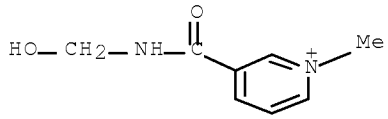
RN 1005-24-9 CAPLUS
CN Pyridinium, 3-(aminocarbonyl)-1-methyl-, chloride (1:1) (CA INDEX NAME)



RN 3106-60-3 CAPLUS
CN Pyridinium, 3-(aminocarbonyl)-1-methyl- (CA INDEX NAME)



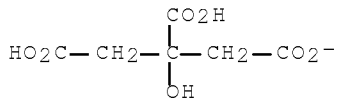
RN 282521-18-0 CAPLUS
CN Pyridinium, 3-[[(hydroxymethyl)amino]carbonyl]-1-methyl- (CA INDEX NAME)



RN 282521-20-4 CAPLUS
CN Pyridinium, 3-(aminocarbonyl)-1-methyl-, 2-hydroxy-1,2,3-propanetricarboxylate (1:1) (CA INDEX NAME)

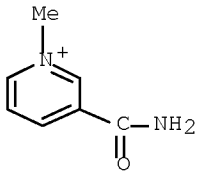
CM 1

CRN 55465-68-4
CMF C6 H7 O7



CM 2

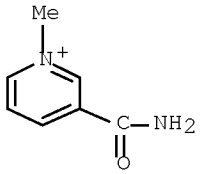
CRN 3106-60-3
CMF C7 H9 N2 O



RN 282521-21-5 CAPLUS
CN Pyridinium, 3-(aminocarbonyl)-1-methyl-, 2-hydroxypropanoate (1:1) (CA INDEX NAME)

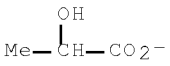
CM 1

CRN 3106-60-3
CMF C7 H9 N2 O

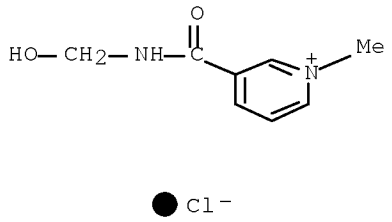


CM 2

CRN 113-21-3
CMF C3 H5 O3



RN 282521-22-6 CAPLUS
CN Pyridinium, 3-[[(hydroxymethyl)amino]carbonyl]-1-methyl-, chloride (1:1) (CA INDEX NAME)



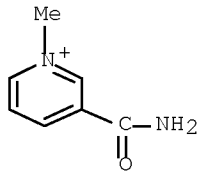
REFERENCE COUNT: 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L50 ANSWER 5 OF 32 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2000:844749 CAPLUS Full-text
DOCUMENT NUMBER: 134:361068
TITLE: Induction of apoptosis in human cancer cells by nicotinic acid-related compounds and their ability for antioxidants
AUTHOR(S): Taguchi, Hiroshi
CORPORATE SOURCE: Laboratory of Biological Chemistry, Faculty of Bioresources, Mie University, Japan
SOURCE: Furi Rajikaru no Rinsho (1999), 14, 23-28
CODEN: FRRIFI
PUBLISHER: Nihon Igakukan
DOCUMENT TYPE: Journal
LANGUAGE: Japanese
ED Entered STN: 05 Dec 2000
AB It was found that picolinic acid, dipicolinic acid, and isonicotinamide strongly induce apoptosis in human acute myelomonocytic leukemia cells, HL-60. Cinchomeronic acid, quinolinic acid, N1-methylnicotinamide, 6-aminonicotinamide, and picolinamide were weak inducers of the apoptosis. After treatments with picolinic acid, dipicolinic acid, and isonicotinamide, apoptosis started within 4 h and it was induced in about 50% of the cells within 8 h. These compds. also induced apoptosis in human chronic myelogenous leukemia cells, K562 and human cervical carcinoma cells, HeLa. However, apoptosis was not induced by these three compds. in quiescent normal human lymphocytes. A wide spectrum caspase inhibitor perfectly prevented DNA fragmentation induced by these compds. But, caspase-1 inhibitor and caspase-3 inhibitor did not block DNA fragmentation. Then the OH radical scavenging effect of nicotinic acid-related compds. was investigated by spin trapping method using DMPO with ESR (ESR). As the result, strong scavenging effect was found with 6-aminonicotinic acid, 6-aminonicotinamide, isonicotinic acid, isonicotinic acid hydrazide, picolinic acid, picolinamide, nicotinic acid hydrazide, etc. The effect of these compds. in HL-60 cells was also investigated after treatment with t-Bu hydroperoxide or hydrogen peroxide by using dichlorofluorescein diacetate as an oxidative state checking probe. As the result, nicotinic acid hydrazide and isonicotinic acid hydrazide were found to be effective for preventing the oxidative state caused by hydrogen peroxide.
CC 1-6 (Pharmacology)
IT 54-85-3, Isonicotinic acid hydrazide 55-22-1, Isonicotinic acid, biological studies 59-67-6D, Nicotinic acid, compds. 89-00-9, Quinolinic acid 98-98-6, Picolinic acid 329-89-5, 6-Aminonicotinamide 490-11-9, Cinchomeronic acid 499-83-2, Dipicolinic acid 553-53-7, Nicotinic acid hydrazide 1452-77-3, Picolinamide 1453-82-3, Isonicotinamide 3106-60-3 3167-49-5, 6-Aminonicotinic acid
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological

study); USES (Uses)
(induction of apoptosis in human cancer cells by nicotinic acid-related compds. and their ability for antioxidants)

IT 3106-60-3
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(induction of apoptosis in human cancer cells by nicotinic acid-related compds. and their ability for antioxidants)

RN 3106-60-3 CAPLUS
CN Pyridinium, 3-(aminocarbonyl)-1-methyl- (CA INDEX NAME)



L50 ANSWER 6 OF 32 MEDLINE on STN
ACCESSION NUMBER: 2008002902 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 17935712
TITLE: Anti-inflammatory effect of 1-methylnicotinamide in contact hypersensitivity to oxazolone in mice; involvement of prostacyclin.
AUTHOR: Bryniarski Krzysztof; Biedron Rafal; Jakubowski Andrzej; Chlopicki Stefan; Marcinkiewicz Janusz
CORPORATE SOURCE: Department of Immunology Jagiellonian University Medical College, Krakow, Poland.
SOURCE: European journal of pharmacology, (2008 Jan 14) Vol. 578, No. 2-3, pp. 332-8. Electronic Publication: 2007-09-26. Journal code: 1254354. ISSN: 0014-2999.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200804
ENTRY DATE: Entered STN: 3 Jan 2008
Last Updated on STN: 4 Apr 2008
Entered Medline: 3 Apr 2008

AB 1-methylnicotinamide (MNA) displays anti-inflammatory effects in patients with contact dermatitis, though the mechanisms involved remain unknown. Herein, we examined the anti-inflammatory effects of MNA and its parent molecule, nicotinamide, in the contact hypersensitivity reaction to oxazolone in CBA/J inbred mice. Feeding mice with MNA or nicotinamide (100 mg/kg, 10 days) resulted in the inhibition of the development of contact hypersensitivity reaction by 37% and 35%, respectively, as assessed by the magnitude of ear swelling. This effect was not associated with changes in the expression of adhesion molecules (CD49d(+) and CD54(+)) on CD4(+) and CD8(+) oxazolone-specific T lymphocytes, the major cell component of an inflammatory infiltrate in contact hypersensitivity reaction. Furthermore, in the adoptive transfer model of contact hypersensitivity reaction, pretreatment of mice (recipients of oxazolone-specific T cells), with MNA, resulted in a remarkable anti-

inflammatory effect (inhibition of contact hypersensitivity reaction by 66%). Interestingly, in the presence of prostanoid IP receptor antagonist R-3-(4-fluoro-phenyl)-2-[5-(4-fluoro-phenyl)-benzofuran-2- ylmethoxycarbonylamino]-propionic acid (RO-3244794) (10 mg/kg) the MNA was inactive. In summary, pretreatment with MNA profoundly attenuated contact hypersensitivity reaction in vivo. In particular, the vessel dependent phase of contact hypersensitivity reaction was affected, in spite of the fact that MNA did not alter the expression of adhesive molecules on oxazolone-specific T lymphocytes. However, the anti-inflammatory action of MNA was completely reversed by the antagonist of prostanoid IP receptor. Accordingly, our results demonstrate for the first time that anti-inflammatory properties of MNA are linked to endothelial, PGI(2)-mediated mechanisms.

CT Check Tags: Male
Adoptive Transfer
Animals
*Anti-Inflammatory Agents: PD, pharmacology
Anti-Inflammatory Agents: TU, therapeutic use
Benzofurans: PD, pharmacology
CD4-Positive T-Lymphocytes: DE, drug effects
CD4-Positive T-Lymphocytes: IM, immunology
CD4-Positive T-Lymphocytes: TR, transplantation
CD8-Positive T-Lymphocytes: DE, drug effects
CD8-Positive T-Lymphocytes: IM, immunology
CD8-Positive T-Lymphocytes: TR, transplantation
Dermatitis, Contact: ET, etiology
Dermatitis, Contact: IM, immunology
Dermatitis, Contact: ME, metabolism
*Dermatitis, Contact: PC, prevention & control
*Dermatologic Agents: PD, pharmacology
Dermatologic Agents: TU, therapeutic use
Disease Models, Animal
*Endothelium, Vascular: DE, drug effects
Endothelium, Vascular: ME, metabolism
*Epoprostenol: ME, metabolism
Integrin alpha4: AN, analysis
Intercellular Adhesion Molecule-1: AN, analysis
Mice
*Niacinamide: AA, analogs & derivatives
Niacinamide: PD, pharmacology
Niacinamide: TU, therapeutic use
Oxazolone
Propionic Acids: PD, pharmacology
Receptors, Prostaglandin: DE, drug effects
Receptors, Prostaglandin: ME, metabolism
Skin: BS, blood supply
*Skin: DE, drug effects
Skin: IM, immunology
Skin: ME, metabolism

L50 ANSWER 7 OF 32 MEDLINE on STN
ACCESSION NUMBER: 2008389237 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 18385449
TITLE: Therapeutic potential of 1-methylnicotinamide against acute gastric lesions induced by stress: role of endogenous prostacyclin and sensory nerves.
AUTHOR: Brzozowski Tomasz; Konturek Peter C; Chlopicki Stefan; Sliwowski Zbigniew; Pawlik Michal; Ptak-Belowska Agata; Kwiecien Slawomir; Drozdowicz Danuta; Pajdo Robert; Slonimska Ewa; Konturek Stanislaw J; Pawlik Wieslaw W

CORPORATE SOURCE: Department of Physiology, Jagiellonian University Medical College, 16 Grzegorzecka St., 31-531 Cracow, Poland.. mpbrzozo@cyf-kr.edu.pl

SOURCE: The Journal of pharmacology and experimental therapeutics, (2008 Jul) Vol. 326, No. 1, pp. 105-16. Electronic Publication: 2008-04-02.
Journal code: 0376362. E-ISSN: 1521-0103.
PUB. COUNTRY: United States
DOCUMENT TYPE: (COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200807
ENTRY DATE: Entered STN: 20 Jun 2008
Last Updated on STN: 31 Jul 2008
Entered Medline: 30 Jul 2008

AB 1-Methylnicotinamide (MNA) is one of the major derivatives of nicotinamide, which was recently shown to exhibit antithrombotic and antiinflammatory actions. However, it is not yet known whether MNA affects gastric mucosal defense. The effects of exogenous MNA were studied on gastric secretion and gastric lesions induced in rats by 3.5 h of water immersion and water restraint stress (WRS) or in rats administered 75% ethanol. MNA [6.25-100 mg/kg intragastrically (i.g.)] led to a dose-dependent rise in the plasma MNA level, inhibited gastric acid secretion, and attenuated these gastric lesions induced by WRS or ethanol. The gastroprotective effect of MNA was accompanied by an increase in the gastric mucosal blood flow and plasma calcitonin gene-related peptide (CGRP) levels, the preservation of prostacyclin (PGI(2)) generation (measured as 6-keto-PGF1alpha), and an overexpression of mRNAs for cyclooxygenase (COX)-2 and CGRP in the gastric mucosa. R-3-(4-Fluoro-phenyl)-2-[5-(4-fluoro-phenyl)-benzofuran-2- ylmethoxycarbonylamino]-propionic acid (RO 324479), which is the selective antagonist of IP/PGI(2) receptors, reversed the effects of MNA on gastric lesions and GBF. MNA-induced gastroprotection was attenuated by suppression of COX-1 [5-(4-chlorophenyl)-1-(4-methoxyphenyl)-3- (trifluoromethyl)-1H-pyrazole; SC-560] and COX-2 [4-(4-methylsulfonylphenyl)-3-phenyl-5H-furan-2-one; rofecoxib] activity, capsaicin denervation, and by the pretreatment with CGRP(8-37) or capsazepine. Addition of exogenous PGI(2) or CGRP restored the MNA-induced gastroprotection in rats treated with COX-1 and COX-2 inhibitors or in those with capsaicin denervation. WRS enhanced MDA content while decreasing superoxide dismutase (SOD) activity in the gastric mucosa, but pretreatment with MNA reversed these changes. MNA exerts potent gastroprotection against WRS damage via mechanisms involving cooperative action of PGI(2) and CGRP in preservation of microvascular flow, antioxidizing enzyme SOD activity, and reduction in lipid peroxidation.

CT Check Tags: Male
Acute Disease
Animals
*Epoprostenol: PH, physiology
Gastric Acid: SE, secretion
Gastric Mucosa: DE, drug effects
Gastric Mucosa: PH, physiology
Gastric Mucosa: SE, secretion
*Neurons, Afferent: DE, drug effects
Neurons, Afferent: PH, physiology
Neurons, Afferent: SE, secretion
*Niacinamide: AA, analogs & derivatives
Niacinamide: PD, pharmacology
Niacinamide: TU, therapeutic use
Rats

Rats, Wistar
*Stomach Ulcer: DT, drug therapy
Stomach Ulcer: ET, etiology
Stress: CO, complications
*Stress: DT, drug therapy

L50 ANSWER 8 OF 32 MEDLINE on STN
ACCESSION NUMBER: 2007526590 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 17641676
TITLE: 1-Methylnicotinamide (MNA), a primary metabolite of nicotinamide, exerts anti-thrombotic activity mediated by a cyclooxygenase-2/prostacyclin pathway.
AUTHOR: Chlopicki S; Swies J; Mogielnicki A; Buczko W; Bartus M; Lomnicka M; Adamus J; Gebicki J
CORPORATE SOURCE: Department of Experimental Pharmacology, Chair of Pharmacology, Jagiellonian University Medical College, Krakow, Poland.. s.chlopicki@cyfronet.krakow.pl
SOURCE: British journal of pharmacology, (2007 Sep) Vol. 152, No. 2, pp. 230-9. Electronic Publication: 2007-07-16. Journal code: 7502536. ISSN: 0007-1188.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: (IN VITRO)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200712
ENTRY DATE: Entered STN: 11 Sep 2007
Last Updated on STN: 19 Dec 2007
Entered Medline: 18 Dec 2007

AB BackgROUND AND PURPOSE: 1-methylnicotinamide (MNA) has been considered to be an inactive metabolite of nicotinamide. Here we assessed the anti-thrombotic activity of MNA in vivo. EXPERIMENTAL APPROACH: Antithrombotic action of MNA was studied in normotensive rats with extracorporeal thrombus formation (thrombolysis), in renovascular hypertensive rats with intraarterial thrombus formation (arterial thrombosis) and in a venous thrombosis model in rats (venous thrombosis). KEY RESULTS: MNA (3-100 mg kg(-1)) induced a dose-dependent and sustained thrombolytic response, associated with a rise in 6-keto-PGF(1alpha) in blood. Various compounds structurally related to MNA were either inactive or weaker thrombolytics. Rofecoxib (0.01-1 mg kg(-1)), dose-dependently inhibited the thrombolytic response of MNA, indomethacin (5 mg kg(-1)) abolished it, while L-NAME (5 mg kg(-1)) were without effect. MNA (3-30 mg kg(-1)) also reduced arterial thrombosis and this effect was abrogated by indomethacin (2.5 mg kg(-1)) as well as by rofecoxib (1 mg kg(-1)). MNA, however, did not affect venous thrombosis. In vitro MNA did not modify platelet aggregation nor induce vasodilation. CONCLUSIONS AND IMPLICATIONS: MNA displayed a profile of anti-thrombotic activity in vivo that surpasses that of closely related compounds. MNA inhibited platelet-dependent thrombosis by a mechanism involving cyclooxygenase-2 and prostacyclin. Our findings suggest that endogenous MNA, produced in the liver by nicotinamide N-methyltransferase, could be an endogenous activator of prostacyclin production and thus may regulate thrombotic as well as inflammatory processes in the cardiovascular system.

CT Check Tags: Male
Animals
Aorta: DE, drug effects
Aorta: PH, physiology
*Cyclooxygenase 2: ME, metabolism
Cyclooxygenase 2 Inhibitors: PD, pharmacology
Epoprostenol: BL, blood

*Epoprostenol: ME, metabolism
*Fibrinolytic Agents: PD, pharmacology
Hypertension: DT, drug therapy
Hypertension: PP, physiopathology
Lactones: PD, pharmacology
Mesenteric Arteries: DE, drug effects
Mesenteric Arteries: PH, physiology
*Niacinamide: AA, analogs & derivatives
Niacinamide: PD, pharmacology
Platelet Aggregation: DE, drug effects
Prostaglandins: BL, blood
Rats
Rats, Wistar
Sulfones: PD, pharmacology
Vasodilation: DE, drug effects
Venous Thrombosis: DT, drug therapy
Venous Thrombosis: PP, physiopathology

L50 ANSWER 9 OF 32 MEDLINE on STN
ACCESSION NUMBER: 2006273353 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 16702628
TITLE: Search for drugs of the combined anti-inflammatory and anti-bacterial properties: 1-methyl-N'-(hydroxymethyl)nicotinamide.
AUTHOR: Adamiec Maciej; Adamus Jan; Ciebiada Ireneusz; Denys Andrzej; Gebicki Jerzy
CORPORATE SOURCE: Chair of Microbiology, Medical University, Hallera 1, PL 90-647 Lodz, Poland.
SOURCE: Pharmacological reports : PR, (2006 Mar-Apr) Vol. 58, No. 2, pp. 246-9.
Journal code: 101234999. ISSN: 1734-1140.
PUB. COUNTRY: Poland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200610
ENTRY DATE: Entered STN: 17 May 2006
Last Updated on STN: 27 Oct 2006
Entered Medline: 26 Oct 2006

AB It has already been reported that 1-methylnicotinamide (MNA+), a primary metabolite of nicotinamide (vitamin B3), possesses remarkable anti-inflammatory properties [3]. This communication shows that 1-methyl-N'-(hydroxymethyl)nicotinamide (MNAF+ can be regarded as MNA+ precursor able to release simultaneously formaldehyde. Therefore, MNAF+ can be viewed as a candidate for drug with the combined anti-inflammatory and anti-bacterial properties.

CT *Anti-Bacterial Agents: CS, chemical synthesis
*Anti-Bacterial Agents: PD, pharmacology
*Anti-Inflammatory Agents: CS, chemical synthesis
*Anti-Inflammatory Agents: PD, pharmacology
Bacteria: DE, drug effects
Drug Design
Formaldehyde: AN, analysis
Indicators and Reagents
Magnetic Resonance Spectroscopy
Microbial Sensitivity Tests
*Niacinamide: AA, analogs & derivatives
Niacinamide: CS, chemical synthesis
Niacinamide: PD, pharmacology

L50 ANSWER 10 OF 32 MEDLINE on STN
ACCESSION NUMBER: 2005523641 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 16197374
TITLE: Topical application of 1-methylnicotinamide in the treatment of rosacea: a pilot study.
AUTHOR: Wozniacka A; Wieczorkowska M; Gebicki J; Sysa-Jedrzejowska A
CORPORATE SOURCE: Department of Dermatology, Medical University of Lodz, Poland.. wozniacka@bmp.net.pl
SOURCE: Clinical and experimental dermatology, (2005 Nov) Vol. 30, No. 6, pp. 632-5.
Journal code: 7606847. ISSN: 0307-6938.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200602
ENTRY DATE: Entered STN: 4 Oct 2005
Last Updated on STN: 7 Feb 2006
Entered Medline: 6 Feb 2006

AB Rosacea is a chronic facial dermatosis with a progressive course, which is characterized by the presence of erythema, papules, pustules, telangiectasias and sebaceous gland hyperplasia. However, the aetiology is still unknown; genetic predisposition, gastrointestinal disorders (Helicobacter pylori), infestations with Demodex folliculorum and environmental stimuli are considered to be involved in the inflammatory process. A metabolite of nicotinamide, 1-methylnicotinamide (MNA(+)), has anti-inflammatory properties, and this is the first study to test the effectiveness of this agent in treating rosacea. In total, 34 patients with rosacea were treated with a gel containing 0.25% MNA(+) as a chloride salt, twice daily for 4 weeks, after which improvement was observed in 26/34 cases. The improvement was good in 9/34 and moderate in 17/34, but no clinical effect was noted in seven subjects. In only one case was skin irritation given as the reason for treatment withdrawal. These results indicate that MNA(+) might be a useful agent for treating rosacea.

CT Check Tags: Female; Male
Administration, Topical
Adult
Chronic Disease
*Dermatologic Agents: AD, administration & dosage
*Facial Dermatoses: DT, drug therapy
Gels
Humans
Middle Aged
Niacinamide: AD, administration & dosage
*Niacinamide: AA, analogs & derivatives
Pilot Projects
*Rosacea: DT, drug therapy

L50 ANSWER 11 OF 32 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1997116614 EMBASE Full-text
TITLE: Hepatic uptake of choline in rat liver basolateral and canalicular membrane vesicle preparations.
AUTHOR: Kwon, Younggil; Lee, Ronald D.; Morris, Marilyn E., Dr. (correspondence)
CORPORATE SOURCE: Department of Pharmaceutics, School of Pharmacy, State Univ. of New York at Buffalo, Amherst, NY, United States.

AUTHOR: Kwon, Younggil
CORPORATE SOURCE: Drug Metabolism Department, Central Research, Pfizer Inc., Groton, CT 06340, United States.
AUTHOR: Lee, Ronald D.
CORPORATE SOURCE: Abbott Laboratories, Drug Metabolism Department, Abbott Park, IL 60064, United States.
AUTHOR: Morris, Marilyn E., Dr. (correspondence)
CORPORATE SOURCE: Department of Pharmaceutics, 527 Hochstetter Hall, SUNY/Buffalo, Amherst, NY 14260, United States.
AUTHOR: Morris, Marilyn E., Dr. (correspondence)
CORPORATE SOURCE: Department of Pharmaceutics, 527 Hochstetter Hall, SUNY, Amherst, NY 14260, United States.
SOURCE: Journal of Pharmacology and Experimental Therapeutics, (Nov 1996) Vol. 279, No. 2, pp. 774-781.
Refs: 44
ISSN: 0022-3565 CODEN: JPETAB
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 12 May 1997
Last Updated on STN: 12 May 1997

AB Choline, an endogenous quaternary ammonium ion, is transported into the liver by both saturable and nonsaturable processes. The objective of the present investigation was to determine the driving force(s) for uptake of choline in rat liver basolateral membrane (bILPM) and canalicular membrane (cLPM) vesicles. Choline is transported into an osmotically sensitive intravesicular space in both bILPM and cLPM. Uptake of [(3)H]choline into both bILPM and cLPM exhibited temperature dependence (0°C vs. 37°C). A valinomycin-induced inside-negative K(+) diffusion potential significantly stimulated initial uptake of [(3)H]choline in both vesicles. Choline uptake in bILPM and cLPM was not stimulated in the presence of an inwardly directed sodium gradient or an outwardly directed H(+) gradient, and ATP did not enhance choline uptake in cLPM. Choline itself and structurally similar derivatives, such as hemicholinium-3 and succinylcholine, inhibited [(3)H]choline uptake 11 to 92% (at 10-fold higher concentrations)in bILPM and cLPM. Other cations, including N(1)- methylnicotinamide, thiamine and d- tubocurarine, and cardioglycosides did not inhibit choline transport in either vesicle preparation. In addition, [(3)H]choline uptake into both bILPM and cLPM was enhanced when vesicles were preloaded with nonradiolabeled choline (trans-stimulation). Kinetic studies indicated that choline was transported into bILPM by both saturable and passive processes and into cLPM predominantly by a saturable process. These results suggest that the transport of choline is likely mediated by a potential-sensitive conductive pathway in both bILPM and cLPM. The electrogenic pathway in cLPM may play a role in the reabsorption of choline from bile.

CT Medical Descriptors:
active transport
animal experiment
article
intrahepatic bile duct
*liver membrane
male
*membrane transport
*membrane vesicle
nonhuman
osmosis
priority journal

rat
transport kinetics
CT Drug Descriptors:
1 methylnicotinamide
*choline: EC, endogenous compound
*hemicholinium 3
*suxamethonium
tubocurarine chloride

L50 ANSWER 12 OF 32 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1995335945 EMBASE Full-text
TITLE: Radical formation site of cerebral complex I and Parkinson's disease.
AUTHOR: Fukushima, T. (correspondence); Tawara, T.; Isobe, A.; Hojo, N.; Shiwaku, K.; Yamane, Y.
CORPORATE SOURCE: Dept. of Environmental Medicine, Shimane Medical University, Enya-Cho 89-1, Izumo 693, Japan.
SOURCE: Journal of Neuroscience Research, (1995) Vol. 42, No. 3, pp. 385-390.
ISSN: 0360-4012 CODEN: JNREDK
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 28 Nov 1995
Last Updated on STN: 28 Nov 1995

AB Paraquat was reduced to the paraquat radical via complex I in bovine cerebral mitochondria and accelerated lipid peroxidation. Thirty-kilodalton subunit of complex I was considered to be the radical formation site, because of its marked destruction by the paraquat radical. The lipid peroxidation by the paraquat radical was suppressed not only by superoxide dismutase (SOD) but also by mannitol. The destruction of complex I subunits via lipid peroxidation must have been caused by the hydroxyl radical which was formed from the superoxide radical. The same phenomenon was observed by using 1-methylnicotinamide (MNA), which contains the same partial structure as paraquat in itself and is metabolized from nicotinamide in a living body. We observed NADH oxidation by MNA via cerebral complex I (Km = 26.3 mM), and MNA destroyed some complex I subunits, especially 30-kilodalton protein. Paraquat might be useful for studying the pathogenesis of Parkinson's disease (PD) in vitro, and MNA is expected to be one of the causal substances of PD from the viewpoint of the oxidative stress theory.

CT Medical Descriptors:
animal tissue
article
cattle
enzyme activity
enzyme substrate complex
lipid peroxidation
nonhuman
oxidation reduction reaction
oxidative stress
*parkinson disease: ET, etiology
pathogenesis
priority journal
CT Drug Descriptors:
1 methylnicotinamide
mannitol

oxygen radical
paraquat
reduced nicotinamide adenine dinucleotide
superoxide dismutase

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ACCESSION NUMBER: 1993005638 EMBASE Full-text
TITLE: A multispecific uptake system for taurocholate, cardiac glycosides and cationic drugs in the liver.
AUTHOR: Steen, H., Dr. (correspondence); Merema, M.; Meijer, D.K.F.
CORPORATE SOURCE: Central Laboratory, Department of Blood Coagulation, Neth. Red Cross Blood Transf. Serv., Plesmanlaan 125, 1066 CX Amsterdam, Netherlands.
SOURCE: Biochemical Pharmacology, (1992) Vol. 44, No. 12, pp. 2323-2331.
ISSN: 0006-2952 CODEN: BCPCA6
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 023 Nuclear Medicine
029 Clinical and Experimental Biochemistry
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 7 Feb 1993
Last Updated on STN: 7 Feb 1993

AB To test the hypothesis of multiplicity in carrier-mediated uptake mechanisms for organic cations in the liver and to study the possible relation with bile acid and cardiac glycoside uptake mechanisms, mutual interaction during uptake of various radiolabeled quaternary amines has been studied in isolated rat hepatocytes. Inhibition patterns at low concentrations (1 μM) of the presumed type I monovalent organic cation tri-n-butylmethylammonium were markedly different from those at relatively high concentrations (25 μM). Both the cardiac glycoside K-strophantoside and the bile acid taurocholate clearly reduced the uptake rate of tri-n-butylmethylammonium at 25 μM whereas these agents completely failed to reduce the uptake at low concentrations of the cation. Subsequently, inhibition of uptake of some multivalent amphipathic organic cations (muscle relaxants) for the type II uptake system was investigated. It was found that the uptake of these muscle relaxants both at tracer concentrations (< 1 μM) and at relatively high concentrations (25 μM) was decreased in the presence of low concentrations of the cardiac glycoside K-strophantoside, while taurocholate only inhibited the uptake at the concentration range >25 μM of the muscle relaxants. Procainamide ethobromide, a typical type I organic cation, did not affect the uptake either at the low or high concentration range of the muscle relaxants. It is concluded that for each of the type I-like compounds and type II-like compounds tested at least two systems are involved in uptake into hepatocytes: tri-n-butylmethylammonium in a concentration range ≤ 1 μM is mainly taken up by the type I uptake system and at concentrations ≥ 25 μM also by system(s) that can be inhibited by taurocholate and K-strophantoside. Bulky amphipathic organic (type II) cations at concentrations < 1 μM are also transported by an uptake system that is inhibitable by cardiac glycosides but not by bile salts. At concentrations > 25 μM these compounds are predominantly accommodated by an uptake system that possibly mediates uptake of both cardiac glycosides and bile acids. This concept was supported by the observation that both type II organic cations and bile salts can inhibit ouabain uptake, while type II organic cations as well as the cardiac glycosides reduce taurocholate uptake rate. The present data

support the idea that the liver seems to be equipped with a 'multispecific' uptake system that transports hydrophobic compounds irrespective of charge, including some type I and type II organic cations at relatively high substrate concentrations.

CT Medical Descriptors:
animal cell
article
*cation transport
controlled study
drug concentration
drug specificity
*drug uptake
*liver cell
nonhuman
priority journal
rat

CT Drug Descriptors:
2beta,16beta dipiperidino 5alpha androstan 3alpha ol acetate
dimethobromide: PK, pharmacokinetics
azidoprocainamide methiodide: PK, pharmacokinetics
*cardiac glycoside: PK, pharmacokinetics
*cation: PK, pharmacokinetics
choline: PK, pharmacokinetics
k strophanthin gamma: PK, pharmacokinetics
n methylnicotinamide: PK, pharmacokinetics
ouabain: PK, pharmacokinetics
procainamide ethobromide: PK, pharmacokinetics
radioisotope
rocuronium: PK, pharmacokinetics
*taurocholic acid: PK, pharmacokinetics
thiamine: PK, pharmacokinetics
tributylmethylammonium: PK, pharmacokinetics
tubocurarine chloride: PK, pharmacokinetics
unclassified drug
vecuronium: PK, pharmacokinetics
verapamil: PK, pharmacokinetics

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ACCESSION NUMBER: 1991183600 EMBASE Full-text
TITLE: Contraluminal p-aminohippurate transport in the proximal tubule of the rat kidney. VII. Specificity: Cyclic nucleotides, eicosanoids.
AUTHOR: Ullrich, K.J. (correspondence); Rumrich, G.; Papavassiliou, F.; Kloss, S.; Fritzsch, G.
CORPORATE SOURCE: Max-Planck-Inst. fur Biophysik, Kennedyallee 70, W-6000 Frankfurt am Main 70, Germany.
SOURCE: Pflugers Archiv European Journal of Physiology, (1991) Vol. 418, No. 4, pp. 360-370.
ISSN: 0031-6768 CODEN: PFLABK
COUNTRY: Germany
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 002 Physiology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 16 Dec 1991
Last Updated on STN: 16 Dec 1991

AB Using the stop-flow peritubular capillary microperfusion method the inhibitory potency (apparent K(i) values) of cyclic nucleotides and prostanoids against

contraluminal p-aminohippurate (PAH), dicarboxylate and sulphate transport was evaluated. Conversely the contraluminal transport rate of labelled cAMP, cGMP, prostaglandin E(2), and prostaglandin D(2) was measured and the inhibition by different substrates was tested. Cyclic AMP and its 8-bromo and dibutyryl analogues inhibited contraluminal PAH transport with an apparatus K(i,PAH) of 3.4, 0.63 and 0.52 mmol/l. The respective apparatus K(i,PAH) values of cGMP and its analogues are with 0.27, 0.04 and 0.05 mmol/l, considerably lower. None of the cyclic nucleotides tested interacted with contraluminal dicarboxylate, sulphate and N(1)-methylnicotinamide transport. ATP, ADP, AMP, adenosine and adenine as well as GTP, GDP, GMP, guanosine and guanine did not inhibit PAH transport while most of the phosphodiesterase inhibitors tested did. Time-dependent contraluminal uptake of [(3)H]cAMP and [(3)H]cGMP was measured at different starting concentrations and showed facilitated diffusion kinetics with the following parameters for cAMP: K(m) = 1.5 mmol/l, J(max) = 0.34 pmol s(-1) cm(-1), r (extracellular/intracellular amount at steady state) = 0.91; for cGMP: K(m) = 0.29 mmol/l, J(max) = 0.31 pmol s(-1) cm(-1), r = 0.55. Comparison of apparatus K(i,cGMP) with apparatus K(i,PAH) of ten substrates gave a linear relation with a ratio of 1.83 ± 0.5. All prostanoids applied inhibited the contraluminal PAH transport; the prostaglandins E(1), F(1α), A(1), B(1), E(2), F(2α), D(2), A(2) and B(2) with an apparatus K(i,PAH) between 0.08 and 0.18 mmol/l. The apparatus K(i) of the prostacyclins 6,15-diketo-13,14-dihydroxy-F(1α) (0.22 mmol/l) and Iloprost (0.17 mmol/l) as well as that of leukotriene, B(4) (0.2 mmol/l) was in the same range, while the apparatus K(i,PAH) of the prostacyclins PGI(2) (0.55 mmol/l), 6-keto-PGF(1α) (0.77 mmol/l), and 2,3-6-keto-PGF(1α) (0.57 mmol/l) as well as that of thromboxane B(2) (0.36 mmol/l) was somewhat higher. None of these prostanoids inhibited contraluminal dicarboxylate transport and only PGB(1), E(2) and D(2) inhibited contraluminal sulphate transport (apparatus K(i,SO(4)(2-)) 5.4, 11.0, 17.9 mmol/l respectively). Contraluminal influx of labelled PGE(2) showed complex transport kinetics with a mixed K(m) = 0.61 mmol/l and J(max) of 4.26 pmol s(-1) cm(-1). It was inhibited by probenecid, sulphate and indomethacin. Contraluminal influx of PGD(2), however, was only inhibited by probenecid. The data indicate that cyclic nucleotides as well as prostanoids are transported by the contraluminal PAH transporter. For prostaglandin E(2) a significant uptake through the sulphate transporter occurs in addition. The hypothesis that prostaglandins as well as 8-bromo and dibutyryl cyclic nucleotides permeate cell membranes by simple diffusion because of their lipid solubility must be considered with reservation.

CT Medical Descriptors:
animal tissue
article
*cell membrane permeability
controlled study
*kidney proximal tubule
male
nonhuman
priority journal
rat

CT Drug Descriptors:
*4 aminohippuric acid: CB, drug combination
*4 aminohippuric acid: DO, drug dose
*4 aminohippuric acid: PK, pharmacokinetics
*8 bromo cyclic amp: CM, drug comparison
*8 bromo cyclic amp: PD, pharmacology
*cyclic amp: CM, drug comparison
*cyclic amp: DO, drug dose
*cyclic amp: PK, pharmacokinetics
*cyclic amp: PD, pharmacology

*cyclic amp derivative: CM, drug comparison
*cyclic amp derivative: PD, pharmacology
*cyclic gmp: CM, drug comparison
*cyclic gmp: DO, drug dose
*cyclic gmp: PK, pharmacokinetics
*cyclic gmp: PD, pharmacology
*cyclic gmp derivative: CM, drug comparison
*cyclic gmp derivative: PD, pharmacology
*enzyme inhibitor: CB, drug combination
*enzyme inhibitor: CM, drug comparison
*enzyme inhibitor: PD, pharmacology
*prostaglandin d2: CM, drug comparison
*prostaglandin d2: PD, pharmacology
*prostaglandin derivative: CM, drug comparison
*prostaglandin derivative: PD, pharmacology
*prostaglandin e2: CM, drug comparison
*prostaglandin e2: PD, pharmacology

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ACCESSION NUMBER: 1989137319 EMBASE Full-text
TITLE: Electron transfer process in cytochrome oxidase after pulse radiolysis.
AUTHOR: Kobayashi, K.; Une, H.; Hayashi, K.
CORPORATE SOURCE: Institute of Scientific and Industrial Research, Osaka University, Osaka 567, Japan.
SOURCE: Journal of Biological Chemistry, (1989) Vol. 264, No. 14, pp. 7976-7980.
ISSN: 0021-9258 CODEN: JBCHA3
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
029 Clinical and Experimental Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 12 Dec 1991
Last Updated on STN: 12 Dec 1991

AB The reduction of bovine heart cytochrome oxidase by the 1- methylnicotinamide (MNA) radical was investigated by the use of pulse radiolysis. With the decay of the MNA radical, the absorption of 445 and 605 nm, a characteristic to ferrous heme a of the oxidase, increased. The kinetic difference spectrum obtained was similar to that of the fully reduced minus the fully oxidized form of the oxidase, and was not different from that obtained in the reaction of the MNA radical with the mixed valence CO complex of the oxidase, where heme a(3) is the CO-bound reduced form with heme a oxidized. This suggests that the absorption changes at 445 and 605 nm arise from the reduction of heme a, not heme a(3). In order to elucidate the contribution of 'visible' copper in this reaction, the absorption of the oxidase in the near-infrared region was measured. A decrease of the 830 nm band due to the reduction of visible copper was detected with a half-life of 5 μs. This absorption change obeyed pseudo-first order kinetics and its rate constant increased with the concentration of the oxidase. This suggests that the absorption change at 830 nm is followed by a bimolecular reaction of the MNA radical with visible copper of the oxidase. After the first phase of the reduction, the return of the 830 nm band corresponding to oxidation of the copper was observed with a half-life of 100 μs. Concomitantly, the absorption at 605 and 445 nm due to the reduction of heme a increased. The rates of oxidation of the copper were identical to those of the reduction of heme a and independent of the oxidase concentration. This suggests that the MNA radical reacts with visible copper of the oxidase with a second order rate constant of 1.5 x 10(9) M(-1) s(-1)

and subsequently the electron flows to heme a by intramolecular electron migration with a first order rate constant of 1.8 x 10(4) s(-1). An activation energy of the intramolecular electron transfer was calculated to be 2.8 kcal/mol in the range 4-33°C.

CT Medical Descriptors:
animal cell
cattle
*heart
nonhuman
priority journal
*pulse radiolysis
CT Drug Descriptors:
*1 methylnicotinamide
*copper
*cytochrome c oxidase
*radical

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ACCESSION NUMBER: 1989022136 EMBASE Full-text
TITLE: Effects of quinidine on the renal tubular and biliary transport of digoxin: In vivo and in vitro studies in the dog.
AUTHOR: Koren, G.; Klein, J.; Giesbrecht, E.; Dayan, R.B.; Soldin, S.; Sellers, E.; MacLeod, S.; Silverman, M.
CORPORATE SOURCE: Division of Clinical Pharmacology, Hospital for Sick Children, University of Toronto, Toronto, Ont. M5G 1X8, Canada.
SOURCE: Journal of Pharmacology and Experimental Therapeutics, (1988) Vol. 247, No. 3, pp. 1193-1198.
ISSN: 0022-3565 CODEN: JPETAB
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 023 Nuclear Medicine
029 Clinical and Experimental Biochemistry
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 12 Dec 1991
Last Updated on STN: 12 Dec 1991

AB Quinidine is known to inhibit the renal clearance of digoxin without affecting glomerular filtration rate. The renal interaction between these drugs was investigated by a combination of in vivo and in vitro methods. The uptake of digoxin by brush border membrane vesicles was not affected by quinidine. Similarly, digoxin did not inhibit the uptake of the cation N-methylnicotinamide by these vesicles and did not alter the binding kinetics of digoxin to the Na(+),K(+)-adenosine triphosphatase by the antiluminal membrane vesicles. By using the in vivo multiple indicator dilution technique transtubular transport of digoxin was documented; renal-artery infusion of quinidine did not affect the recovery of digoxin in the renal vein or urine. Clearance studies documented that the decrease in the renal clearance of digoxin is paralleled by a significant fall in renal blood flow evidenced by a decrease in p-aminohippuric acid clearance. It is concluded that quinidine inhibits the renal excretion of digoxin not by competition at the tubular cell membrane level, but rather by decreasing renal blood flow. A parallel decrease in biliary clearance of digoxin is documented and may suggest a similar mechanism.

CT Medical Descriptors:
animal cell

animal experiment
*brush border vesicle
dog
*drug bile level
drug binding
*drug clearance
*drug transport
female
intraarterial drug administration
intravenous drug administration
*kidney tubule
male
nonhuman
priority journal

CT Drug Descriptors:
4 aminohippuric acid
cimetidine
*digoxin: CR, drug concentration
*digoxin: IT, drug interaction
*digoxin: PK, pharmacokinetics
n methylnicotinamide
*quinidine: IT, drug interaction
radioisotope

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ACCESSION NUMBER: 1987057725 EMBASE Full-text
TITLE: Transport of cimetidine by the rat choroid plexus in vitro.
AUTHOR: Suzuki, H.; Sawada, Y.; Sugiyama, Y.; et. al.
CORPORATE SOURCE: Faculty of Pharmaceutical Sciences, University of Tokyo, Hongo, Bunkyo-ku, Tokyo 113, Japan.
SOURCE: Journal of Pharmacology and Experimental Therapeutics, (1986) Vol. 239, No. 3, pp. 927-935.
ISSN: 0022-3565 CODEN: JPETAB
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 030 Clinical and Experimental Pharmacology
037 Drug Literature Index
048 Gastroenterology
008 Neurology and Neurosurgery
LANGUAGE: English
ENTRY DATE: Entered STN: 11 Dec 1991
Last Updated on STN: 11 Dec 1991

AB To characterize the transport system of cimetidine, an organic cation, in the blood-cerebrospinal fluid barrier, the accumulation of cimetidine by the isolated rat choroid plexus was examined. Accumulation of cimetidine was against a concentration gradient via a saturable process (K(m) = 53 μM, V(max) = 12 nmol/ml/min) that was inhibited by sulfhydryl reagents (p-hydroxcymercuribenzoate), metabolic inhibitors (KCN and 2,4-dinitrophenol) and hypoetermia (Q(10) = 4.5), but did not require inward Na(+) gradient. Organic cations such as (1)N- methylnicotinamide, tetraethylammonium, choline, histamine and creatinine did not affect the accumulation of cimetidine at the concentration of 1 mM. Cimetidine did not affect the accumulation of tetraethylammonium. More lipophilic cations such as quinidine and quinine inhibited not only the accumulation of cimetidine but also that of an organic anion, benzylpenicillin, although the inhibitory mechanisms are not known. One millimolar of organic anions, such as 5- hydroxyindoleacetic acid, p-aminohippuric acid, homovanillic acid, salicyclic acid and benzylpenicillin, inhibited the accumulation of cimetidine. Furthermore, the accumualtion of organic anions (benzylpenicilln and salicylic acid) showed saturability and

was inhibited by cimetidine. Cimetidine and the organic anions thus showed a mutual inhibition. Oligopeptides also inhibited the accumualtion of cimetidine. These findings suggests that cimetidine transport in the choroid plexus is via carrier-mediated active transport process, but does not require inward Na(+) gradient. This transport is inhibited by several compounds with different properties like oligopeptides, lipophilic cations and organic anions, although the inhibitory mechanism is not known.

CT Medical Descriptors:
animal cell
article
*autoradiography
*blood brain barrier
central nervous system
*choroid plexus
*drug accumulation
*drug transport
nervous system
nonhuman
peripheral vascular system
pharmacokinetics
rat

CT Drug Descriptors:
*anion
*cation
*cimetidine
*cimetidine h 3
*polypeptide
radioisotope
unclassified drug

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ACCESSION NUMBER: 1985228980 EMBASE Full-text
TITLE: Effect of cisplatin on organic ion transport in membrane vesicles from rat kidney cortex.
AUTHOR: Williams, P.D.; Hottendorf, G.H.
CORPORATE SOURCE: Experimental Toxicology Department, Bristol-Myers Co, Pharmaceutical Research and Development Division, Syracuse, NY 13221-4755, United States.
SOURCE: Cancer Treatment Reports, (1985) Vol. 69, No. 7-8, pp. 875-880.
ISSN: 0361-5960 CODEN: CTRRDO
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
028 Urology and Nephrology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Dec 1991
Last Updated on STN: 10 Dec 1991

AB Purified renal membrane vesicles were utilized to gain indirect information regarding the renal handling of cisplatin. The effects of cisplatin on prototypical organic anion (p-aminohippurate, PAH) and cation (N(1)-methylnicotinamide; tetraethylammonium, TEA) transport in brush border and basolateral membrane vesicles prepared from rat kidney cortex were observed. While cisplatin inhibited organic cation transport (N(1)-methylnicotinamide; TEA) in brush border and basolateral membranes, no interaction with the organic anion (p-aminohippurate) system was observed. Kinetic analyses revealed that cisplatin is a competitive inhibitor of TEA transport in brush

border membranes with a k(i) of 0.12 mM. While the relationship between organic cation transport inhibition and cisplatin nephrotoxicity is unknown, it may suggest that the cisplatin complex itself is transported into the kidney by the organic cation system. The reported effect of the organic anion, probenecid, on the renal handling of cisplatin is discussed in light of these results.

CT Medical Descriptors:
*adverse drug reaction
animal cell
article
*cancer chemotherapy
*drug efficacy
*drug sensitivity
*ion transport
kidney
*kidney cortex
membrane vesicle
nonhuman
peripheral vascular system
priority journal
rat
therapy
topical drug administration

CT Drug Descriptors:
1 methylnicotinamide h 3
4 aminohippuric acid h 3
*cisplatin
*piperphenamine
*probenecid
radioisotope
tetraethylammonium h 3
unclassified drug

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ACCESSION NUMBER: 1985081581 EMBASE Full-text
TITLE: Estimation of renal blood flow by use of endogenous N(1)-methylnicotinamide in rats.
AUTHOR: Shim, C.K.; Sawada, Y.; Iga, T.; Hanano, M.
CORPORATE SOURCE: Faculty of Pharmaceutical Sciences, University of Tokyo, Hongo, Bunkyo-ku, Tokyo 113, Japan.
SOURCE: Journal of Pharmacobio-Dynamics, (1985) Vol. 8, No. 1, pp. 20-24.
ISSN: 0386-846X CODEN: JOPHDQ
COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
028 Urology and Nephrology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Dec 1991
Last Updated on STN: 10 Dec 1991

CT Medical Descriptors:
*acute kidney failure
animal experiment
article
*drug efficacy
*drug interaction
kidney

*kidney blood flow
*kidney failure
nonhuman
peripheral vascular system
rat
CT Drug Descriptors:
*1 methylnicotinamide
4 aminohippuric acid
*folic acid
gentamicin
glycerol
inulin h 3
radioisotope
salicylate sodium
salicylic acid
unclassified drug
uranium
uranium nitrate

L50 ANSWER 20 OF 32 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1984163298 EMBASE Full-text
TITLE: Studies on nifurtimox nitroreductase activity in liver and other rat tissues.
AUTHOR: Masana, M.; De Toranzo, E.G.D.; Castro, J.A.
CORPORATE SOURCE: Centro de Investigaciones Toxicologicas (Ceitox) - Citefa/Conicet, Pcia. de Buenos Aires, Buenos Aires, Argentina.
SOURCE: Archives Internationales de Pharmacodynamie et de Therapie, (1984) Vol. 270, No. 1, pp. 4-10.
ISSN: 0003-9780 CODEN: AIPTAK
COUNTRY: Belgium
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical and Experimental Biochemistry
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
048 Gastroenterology
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Dec 1991
Last Updated on STN: 10 Dec 1991
AB Rat liver microsomes exhibit nifutimox (NFX) nitroreductase activity, which is mostly NADPH-dependent and is completely abolished by heating and under an atmosphere of air. Pure carbon monoxide inhibits for 28% microsomal NFX nitroreductase activity while FAD 1 mM significantly enhances it. Smaller activities than in liver were found in brain, small intestine, testes, lung and heart. Rat liver cytosol also showed NFX nitroreductase activity using either hypoxanthine or N- methylnicotinamide as substrates. These activities were inhibited by allopurinol or menadione respectively. Results suggest that cytochrome P-450, NADPH cytochrome c reductase, xanthinoxidase and aldehyde oxidase are able to reduce NFX nitrogroups in rat liver and other tissues.
CT Medical Descriptors:
animal cell
cytosol
drug administration
*drug efficacy
*drug inhibition
*drug metabolism
etiology
*liver
methodology

microsome
nonhuman
rat
therapy
CT Drug Descriptors:
allopurinol
*enzyme
hypoxanthine
menadione
*mixed function oxidase
n methylnicotinamide
*nifurtimox
*nitroreductase

L50 ANSWER 21 OF 32 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1983064892 EMBASE Full-text
TITLE: Factors influencing the efflux of hepatic glutathione into bile in rats.
AUTHOR: Kaplowitz, N.; Eberle, D.E.; Petrini, J.; et. al.
CORPORATE SOURCE: Gastroenterol. Sect., Med. Res. Serv., VA Wadsworth Hosp., Los Angeles, CA 90073, United States.
SOURCE: Journal of Pharmacology and Experimental Therapeutics, (1983) Vol. 224, No. 1, pp. 141-147.
ISSN: 0022-3565 CODEN: JPETAB
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical and Experimental Biochemistry
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
048 Gastroenterology
052 Toxicology
LANGUAGE: English
ENTRY DATE: Entered STN: 9 Dec 1991
Last Updated on STN: 9 Dec 1991

AB A major factor in hepatic glutathione (GSH) homeostasis appears to be efflux of GSH from the liver into both bile and blood. In order to gain insight into the factors which influence hepatic GSH efflux into bile, the effects of in vivo perturbations of bile flow and hepatic GSH concentration were determined in the rat. GSH efflux into bile was not enhanced by stimulation of bile secretion by taurocholate or sulfobromophthalein-GSH conjugate. Moreover, sulfobromophthalein-GSH, probenecid and N'-methylnicotinamide, substances excreted into bile by carrier-mediated processes, had no effect on bile GSH excretion. Treatment with diethyl maleate, azathioprine and acetaminophen to deplete or 3-methylcholanthrene to increase hepatic GSH concentration revealed a direct relationship between bile GSH output and hepatic GSH concentration. The effect of phenobarbital treatment on bile GSH efflux was conspicuously different from 3-methylcholanthrene; both caused an approximate 30% increase in hepatic GSH concentration but only phenobarbital markedly enhanced bile GSH output (approximately 250%). This effect of phenobarbital on bile GSH was manifest over a wide range of hepatic GSH produced in response to varying doses of acetaminophen. Oxidized GSH excretion in bile was not affected by cholephilic substances or inducing agents. By examining the time course of the effects of a single dose of phenobarbital, the enhanced bile GSH excretion could be dissociated from phenobarbital-induced increase in bile secretion or hepatic GSH. Sinusoidal GSH efflux in the in situ perfused rat liver and plasma GSH concentration in the hepatic vein in vivo were unaffected by phenobarbital. In conclusion, canalicular GSH excretion is a contributing factor to hepatic GSH homeostasis. This process is quantitatively related to hepatic GSH content but is not influenced by bile secretory rates or the known

carrier-mediated transport processes. Inasmuch as phenobarbital treatment enhanced only bile GSH efflux, the canalicular route for GSH excretion may be selectively altered by certain xenobiotics, thereby affecting GSH homeostasis.

CT Medical Descriptors:
animal experiment
article
*bile
*drug efficacy
drug transport
*liver
nonhuman
pharmacokinetics
rat
CT Drug Descriptors:
*3 methylcholanthrene
*azathioprine
*glutathione
*maleic acid diethyl ester
*malic acid diethyl ester
*paracetamol
*phenobarbital
*taurocholic acid
unclassified drug

L50 ANSWER 22 OF 32 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1981023581 EMBASE Full-text
TITLE: Effects of excess leucine on growth and tryptophan and niacin metabolism in rats.
AUTHOR: Ohguri, S.
CORPORATE SOURCE: Dept. Maternal Child Hlth, Inst. Publ. Hlth, Tokyo 108, Japan.
SOURCE: Journal of Nutritional Science and Vitaminology, (1980) Vol. 26, No. 2, pp. 141-160.
ISSN: 0301-4800 CODEN: JNSVA5
COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 037 Drug Literature Index
LANGUAGE: English
ENTRY DATE: Entered STN: 9 Dec 1991
Last Updated on STN: 9 Dec 1991
CT Medical Descriptors:
animal experiment
body weight
drug blood level
drug urine level
heart
liver
n methyl 2 pyridone 5 carboxamide
*nutrition
oral drug administration
organ
*pellagra
pharmacokinetics
preliminary communication
rat
urinary excretion
*vitamin metabolism
CT Drug Descriptors:
5 hydroxyindoleacetic acid

*amino acid
*leucine
n methylnicotinamide
*nicotinic acid
quinolinic acid
*tryptophan

L50 ANSWER 23 OF 32 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1979234232 EMBASE Full-text
TITLE: Drug entry into the brain.
AUTHOR: Rapoport, S.I.; Ohno, K.; Pettigrew, K.D.
CORPORATE SOURCE: Lab. Neurosci., Nat. Inst. Aging, Gerontol. Res. Cent., Baltimore City Hosp., Baltimore, Md. 21224, United States.
SOURCE: Brain Research, (1979) Vol. 172, No. 2, pp. 354-359.
ISSN: 0006-8993 CODEN: BRREAP
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 002 Physiology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: English
AB The selective permeability of the blood-brain barrier to lipid-soluble drugs qualitatively explains different rates of drug passage from plasma to spinal fluid and brain, but the pharmacokinetics of plasma-brain exchange have not been quantified. This report establishes an empirical quantitative relation between cerebrovascular permeability and the octanol-water partition coefficient, and thereby makes it possible to estimate brain accumulation of a drug from the partition coefficient and the history of the plasma concentration. Such a relation is useful because it is often difficult in man to relate the action of a centrally-acting drug or its derivatives to plasma or cerebrospinal fluid concentrations. Plasma concentrations do not represent brain concentrations because the continuous cerebrovascular endothelium interferes with plasma-brain exchange. Spinal fluid concentration may be lower than brain concentration if a drug is very lipid-soluble and accumulates in brain lipids, or higher if, like some proteins, it enters the spinal fluid at the choroid plexus.
CT Medical Descriptors:
animal experiment
article
*blood brain barrier
*brain blood flow
*brain cerebrospinal fluid barrier
central nervous system
*choroid plexus
drug distribution
oral drug administration
peripheral vascular system
rat
CT Drug Descriptors:
*acetamide
*arabinose
*caffeine
*curare
*erythritol
*ethylene glycol
*formamide
*glycerol
*mannitol
*methotrexate

*n methylnicotinamide
*phenazone
*propylene glycol
radioisotope
*sucrose
sucrose c 14
*tetrylammonium
*thiourea
unclassified drug
*urea

L50 ANSWER 24 OF 32 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1978343119 EMBASE Full-text
TITLE: Induction of ornithine decarboxylase by nicotinamide, 5-methylnicotinamide and thymidine.
AUTHOR: Minaga, T.; Piper, W.N.; Kun, E.
CORPORATE SOURCE: Dept. Pharmacol., Univ. California, San Francisco, Calif. 94143, United States.
SOURCE: Federation Proceedings, (1978) Vol. 37, No. 3, pp. No. 2017.
ISSN: 0014-9446 CODEN: FEPRA7
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
LANGUAGE: English
CT Medical Descriptors:
animal experiment
*dose response
*drug comparison
drug response
*enzyme induction
*heart
intraperitoneal drug administration
*liver
*rat
CT Drug Descriptors:
*5 methylnicotinamide
*cycloheximide
*nicotinamide
*ornithine decarboxylase
*putrescine
*thymidine
unclassified drug

L50 ANSWER 25 OF 32 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1978242723 EMBASE Full-text
TITLE: Effect of niacin and thiamine deficiency on tolerance of the myocardium to hypoxia.
AUTHOR: Salzsieder, K.H.; Bing, O.H.L.; Abelmann, W.H.
CORPORATE SOURCE: Dept. Med., Harvard Med. Sch., Boston, Mass., United States.
SOURCE: Clinical Research, (1977) Vol. 25, No. 5, pp. 654a.
ISSN: 0009-9279 CODEN: CLREAS
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
LANGUAGE: English
CT Medical Descriptors:

*anaerobic metabolism
*heart muscle
*hypoxia
in vitro study
*rat
theoretical study
*vitamin deficiency
CT Drug Descriptors:
*1 methylnicotinamide
*nicotinic acid
*thiamine

L50 ANSWER 26 OF 32 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1977196459 EMBASE Full-text
TITLE: Metabolic response of humans to ingestion of nicotinic acid and nicotinamide.
AUTHOR: Mrochek, J.E.; Jolley, R.L.; Young, D.S.; Turner, W.J.
CORPORATE SOURCE: Oak Ridge Nat. Lab., Oak Ridge, Tenn. 37830, United States.
SOURCE: Clinical Chemistry, (1976) Vol. 22, No. 11, pp. 1821-1827. ISSN: 0009-9147 CODEN: CLCHAU
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical and Experimental Biochemistry
037 Drug Literature Index
LANGUAGE: English
AB The identification of nicotinamide-N(1)-oxide as a metabolite in the urine of a schizophrenic patient prompted a study of the relative metabolism of nicotinic acid and nicotinamide in mental patients and healthy volunteers. Metabolites quantified included N(1)-methyl-2-pyridone-5-carboxamide, N(1)-methyl-4-pyridone- 3-carboxamide, N(1)-methylnicotinamide, nicotinuric acid, and nicotinamide-N(1)-oxide. More of most of these metabolites evidently was excreted after nicotinamide ingestion than after nicotinic acid. At the highest doses (3000 mg/day), the relative proportions of these metabolites in the urine were changed. There were only slight differences between healthy individuals and mental patients in the quantities of metabolites excreted, and no statistically significant trends were noted.
CT Medical Descriptors:
*adverse drug reaction
*dose response
drug blood level
*drug excretion
*drug metabolism
drug urine level
*heart disease
in vitro study
*liquid chromatography
normal human
oral drug administration
pharmacokinetics
*psychosis
*schizophrenia
theoretical study
*urine
CT Drug Descriptors:
*nicotinamide
*nicotinic acid

L50 ANSWER 27 OF 32 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1977028886 EMBASE Full-text

TITLE: Binding of organic compounds to rat liver and lung.
AUTHOR: Ludden, T.M.; Schanker, L.S.; Lanman, R.C.
CORPORATE SOURCE: Dept. Pharmacol., Univ. Missouri, Kansas City, Mo. 64110, United States.
SOURCE: Drug Metabolism and Disposition, (1976) Vol. 4, No. 1, pp. 8-16.
ISSN: 0090-9556 CODEN: DMDSAI
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: English

AB The binding of various radioisotopically labeled organic compounds to rat liver and lung was investigated in vitro. Pieces of rat lung and slices of rat liver were incubated at 37°C under a nitrogen atmosphere in a modified Krebs Ringer phosphate solution (pH 7.4) containing the compound to be studied. Of the neutral compounds investigated, digitoxin, digoxin, and dexamethasone were highly bound to both liver and lung tissue, whereas the degree of binding of amitrole, erythritol, and ouabain was 20% or less. The weak acids which were bound to the greatest extent in both liver and lung were phenobarbital, pentobarbital, and diphenylhydantoin. Barbital was poorly bound, and there was no evidence for the binding of 5,5 dimethyloxazolidine 2,4 dione or p aminohippuric acid in either tissue. Binding of the cardiac glycosides and the barbiturates directly paralleled their lipid solubilities. The degree of binding of neutral compounds and weak acids to lung and liver tissue did not vary greatly with concentration, even though broad concentration ranges were studied. This was also true of the weak base morphine. On the other hand, the binding to liver and lung of the organic bases nicotine, pilocarpine, d amphetamine, lidocaine, erythromycin, and chloroquine, did vary with concentration. The quaternary ammonium compound decamethonium was bound only to liver, and this binding also varied with concentration. Two additional quaternary ammonium compounds, tetraethylammonium and N(1) methylnicotinamide, were not significantly bound to either tissue. Comparisons on the basis of equal content of solids revealed that the binding of diverse organic compounds in liver is greater than or equal to that in lung.

CT Medical Descriptors:
article
*drug binding
in vitro study
*liver
*lung
*pH
*rat
theoretical study

CT Drug Descriptors:
*4 aminohippuric acid c 14
amitrole
*barbituric acid derivative
*chloroquine c 14
*decamethonium c 14
*dexamethasone h 3
*dexamphetamine
*digitoxin h 3
*digoxin h 3
*dimethyltubocurarine c 14
*erythritol c 14
*erythromycin c 14
*lidocaine c 14
*morphine c 14
*nicotine c 14

*ouabain h 3
*pentobarbital c 14
*phenytoin c 14
*pilocarpine
radioisotope
unclassified drug

L50 ANSWER 28 OF 32 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1976010237 EMBASE Full-text
TITLE: [Absorption and secretion of drugs by the mucosal epithelia of the gastrointestinal tract].
RESORPTION UND SEKRETION VON ARZNEISTOFFEN DURCH DIE MUKOSAEPITHELIEN DES GASTROINTESTINALTRAKTES.
AUTHOR: Lauterbach, F.
CORPORATE SOURCE: Inst. Pharmakol. Toxikol., Ruhr Univ., Bochum, Germany.
SOURCE: Arzneimittel-Forschung/Drug Research, (1975) Vol. 25, No. 3 A, pp. 479-488.
ISSN: 0004-4172 CODEN: ARZNAD
DOCUMENT TYPE: Journal
FILE SEGMENT: 030 Clinical and Experimental Pharmacology
037 Drug Literature Index
048 Gastroenterology
LANGUAGE: German

AB The intestinal absorption of numerous drugs can be described correctly by the principle of nonionic diffusion, i.e. by diffusion of their uncharged, lipophilic moieties across the membranes of the mucosal epithelium. Evidence for participation of transport mechanisms in absorption of drugs was recently obtained. An experimental procedure was developed which uses the isolated mucosal epithelium of guineapig jejunum as a separating membrane between 2 flux chambers. Diffusing drugs such as hydrocortisone permeate the epithelium at equal rates in both directions (flux ratio = 1). Permeation rate and relative uptake are independent of the concentration offered. Hydrocortisone uptakes from the lumen and blood sides of the preparation are identical. Anaerobiosis does not influence permeation and uptake. The permeation behavior of cardiac glycosides, quaternary ammonium compounds and strongly acidic drugs is described by typical examples. Drugs representative of these classes permeate the mucosa faster from the blood side to the lumen side than in the absorptive direction (flux ratio <1). The rate of permeation from blood to lumen side shows pronounced concentration dependence. Relative uptake from the blood side is higher than that from the lumen side and decreases with increasing drug concentration. After preloading the tissue with digoxin from the blood side, the glycoside is released preferentially to the lumen side. In anaerobiosis the flux ratio equals 1, the uptake from the lumen side is increased and the efflux to the lumen side is reduced. Results obtained so far demonstrate that the intestinal transport mechanisms previously proposed for cardiac glycosides, quaternary ammonium bases and organic acids are in reality secretory mechanisms which are able to transport such drugs against a concentration gradient from the blood into the intestinal lumen. Experiments with the isolated mucosa indicate that transport mechanisms have to be located in series in the lumenal and basolateral membranes of the mucosal cell, which are paralleled by diffusional pathways. The active secretory mechanisms for cardiac glycosides and quaternary ammonium compounds could be substantiated in vivo. After i.v. administration compounds of both classes are concentrated in the intestinal lumen well at above the serum level in guinea pig and rat. Establishment of an equilibrium between concentrations in blood and lumen were demonstrated to be the cause of the previously observed standstill in absorption of quaternary ammonium compounds despite considerable amounts of unabsorbed drug. The results described point to the gut as a third excretory organ besides liver and kidney. Its secretory

mechanisms, which might interfere with the absorption of drugs either by mediation of drug permeation in the reverse (absorption) direction or by resecretion of substance just absorbed, offer an explanation for numerous hitherto incomprehensible peculiarities in the absorption behavior of drugs. Nonionic diffusion is only one possibility for drugs to cross the intestinal epithelium; a second one is the permeation by specific transport mechanisms.

CT Medical Descriptors:
*diffusion
*drug absorption
drug blood level
*drug mechanism
*gastrointestinal mucosa
*gastrointestinal tract
*intestine
*intestine absorption
*intestine mucosa
intravenous drug administration
*mucosa
*pH
pharmacokinetics

CT Drug Descriptors:
*1 methylnicotinamide
*benzomethamine
*carboxylic acid
*cardiac glycoside
*convallaria glycoside
*digoxin
*digoxin h 3
*drug
*hydrocortisone
*inulin h 3
*methylscopolamine
*phenolsulfonphthalein
*quaternary ammonium derivative
radioisotope
*sulfanilic acid s 35
*tetrylammonium bromide
unclassified drug

L50 ANSWER 29 OF 32 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 1977132358 EMBASE Full-text
TITLE: Studies on the effect of nicotinamide and ethylnicotinic acid on cyclic AMP phosphodiesterase from rat liver.
AUTHOR: Hoshi, Y.
CORPORATE SOURCE: Dept. Med. Chem., Osaka Med. Coll., Takatsuki City, Japan.
SOURCE: Bulletin of the Osaka Medical School, (1975) Vol. 21, No. 2, pp. 77-91.
ISSN: 0030-6142 CODEN: BUOSA5
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical and Experimental Biochemistry
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: English

CT Medical Descriptors:
article
*brain
drug comparison
drug response
*enzyme inhibition

*heart
*intestine
intraperitoneal drug administration
*kidney
*liver
*metabolism
*pharmacokinetics
*rat
*spleen
theoretical study
CT Drug Descriptors:
*3 acetylpyridine
*3 pyridinesulfonic acid
*6 aminonicotinamide
*benzamide
*cyclic AMP
*cyclic AMP phosphodiesterase
*liver enzyme
*methylnicotinamide
*nicotinamide
*nicotinamide derivative
*nicotinic acid
*nicotinic acid derivative
*nicotinic acid ethyl ester
*nikethamide
*papaverine
*phosphodiesterase
*pyridine
*theophylline
unclassified drug

L50 ANSWER 30 OF 32 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1976080331 EMBASE Full-text
TITLE: Organic ion transport and O(2) consumption by the non filtering kidney (NFK).
AUTHOR: Bailie, M.D.; Corsini, W.A.; Johnson, J.T.; et. al.
CORPORATE SOURCE: Coll. Hum. Med., Mich. State Univ., East Lansing, Mich., United States.
SOURCE: Clinical Research, (1975) Vol. 23, No. 3, pp. 355A.
ISSN: 0009-9279 CODEN: CLREAS
DOCUMENT TYPE: Journal
FILE SEGMENT: 002 Physiology
028 Urology and Nephrology
LANGUAGE: English
AB The non filtering kidney (NFK) has been used to study the control of renin secretion. Since preparation involves occlusion of the ureter and 2 hr of ischemia questions have arisen concerning the viability of the organ. To investigate the metabolic state of the NFK, the authors utilized the renal cortical slice technique of Cross and Taggart with para aminohippurate (PAH) and N methylnicotinamide (NMN) as prototypes of organic anion and cation transport, respectively. Renal cortical slices were prepared from the NFK and contralateral control kidney of dogs. Oxygen consumption was determined using a Gilson differential respirometer. Accumulation of PAH or NMN was determined as the slice/medium ratio (S/M) after 90 min incubating at 25°C. Portions of the kidneys were fixed for examination by light microscopy. In the NFK, tissue was obtained from areas which grossly appeared normal and areas which were obviously abnormal. As shown in a table, PAH and NMN accumulation were similar in cortex obtained from control kidneys and normal areas of the NFK but were less than in the abnormal appearing tissue. Additional experiments

demonstrate acetate stimulation of PAH uptake in control kidney slices but not in either normal or abnormal slices from NFK. O(2) consumption was significantly less than control in the slices from all areas of the NFK. It is concluded that the NFK is metabolically active. However, there are quantitative differences which may be related to the degree of tissue necrosis.

CT Medical Descriptors:
dog
*glomerulus filtration rate
in vitro study
*kidney
*kidney failure
*oxygen consumption
theoretical study

L50 ANSWER 31 OF 32 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 1974080064 EMBASE Full-text
TITLE: [The efficacy of nicotinic acid in maize or oat diets]. EFFICACITE DE LA NIACINE DANS LES RATIONS A BASE DE MAIS OU D'AVOINE.
AUTHOR: Adrian, J.
CORPORATE SOURCE: Cent. Rech. Nutrit., CNRS, Bellevue, France.
SOURCE: International Journal for Vitamin and Nutrition Research, (1973) Vol. 43, No. 3, pp. 327-338.
ISSN: 0300-9831 CODEN: IZVIAK
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
029 Clinical and Experimental Biochemistry
LANGUAGE: French

AB Nicotinic acid efficacy was evaluated in maize or oat diets with or without supplementation with nicotinic acid, tryptophan, lysine or threonine. Nicotinic acid was determined in serum, liver and urine; urinary N methylnicotinamide was also determined. The 2 cereals have a comparable nicotinic acid equivalent content; maize contains more nicotinic acid, oat more tryptophan. For growing rats, the oat nicotinic acid equivalents were more efficient than those of maize. A single addition of nicotinic acid or tryptophan did not render the maize diet adequate. Tryptophan made the oat diet adequate but nicotinic acid did not. For the nicotinic acid of cereal diets to become fully efficient, the amino acid deficiencies must first be controlled. For adult rats, the maize diet was improved by nicotinic acid and even more by tryptophan. These observations are not in agreement with those made on the growing rats. In rats, nicotinic acid or tryptophan efficiency depends on protein quality of the diet and on nitrogen requirement of the animal. The better protein quality of oat permits a better utilization of nicotinic acid than in maize. Addition of nicotinic acid to maize diet is more efficient for adult than for the growing rats, since cereals are more suited to satisfy the adult nitrogen requirement. Based on a conversion rate of 60:1, the tryptophan in cereal diets appears to be more efficient than its nicotinic acid equivalent.

CT Medical Descriptors:
*cereal
*diet
drug blood level
*drug efficacy
drug urine level
*feeding behavior
*heart ventricle activation
*liver
*maize

*oat
oral drug administration
rat
*serum
theoretical study
*urine
CT Drug Descriptors:
*nicotinic acid

L50 ANSWER 32 OF 32 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 1974015191 EMBASE Full-text
TITLE: Congenital brain cysts in infancy: diagnosis, treatment, and followup.
AUTHOR: Shurtleff, D.B.; Eliason, B.C.; Oakland, J.A.
CORPORATE SOURCE: Div. Congen. Defects, Dept. Ped., Univ. Washington, Seattle, Wash. 98195, United States.
SOURCE: Teratology, (1973) Vol. 7, No. 2, pp. 183-190.
ISSN: 0040-3709 CODEN: TJADAB
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 008 Neurology and Neurosurgery
014 Radiology
007 Pediatrics and Pediatric Surgery
021 Developmental Biology and Teratology
022 Human Genetics
LANGUAGE: English
AB Eleven cases of congenital brain cysts - 2 encephaloclastic intracerebral, 5 noncommunicating pia arachnoid, and 4 either communicating pia arachnoid or intracranial meningoencephalocele congenital type cysts - were diagnosed in infancy based on suspicious cephalomegaly, skull asymmetry, and abnormal transillumination. Complete diagnosis differentiated primary congenital cysts from trauma and infection, both of which complicate congenital cysts as well as being etiologic for brain cyst formation. Early diagnosis coupled with surgical exploration and cerebrospinal fluid shunting have allowed seven to live in a functional, socially acceptable way to the age of 1 1/2 to 12 1/2 yr. Two of the remaining 4 are now dead, and 2 are severely retarded. Retardation was due to congenital malformation and a shunt obstruction. Death resulted from infectious epiglottis and cardiac arrest during cystostomy.
CT Medical Descriptors:
article
*brain arachnoid cyst
*brain cyst
*cerebrospinal fluid shunting
*congenital malformation
*cyst
*diagnostic error
*diagnostic imaging
*encephalomeningocele
*hydrocephalus
*mental deficiency
*pneumoencephalography
*porencephaly
*skull
CT Drug Descriptors:
*n [2 [3,4 bis(tert butyryl)phenyl]ethyl] 1,4 dihydro 1 methylnicotinamide
unclassified drug

L51 ANSWER 1 OF 14 CAPLUS COPYRIGHT 2008 ACS on STN DUPLICATE 1
ACCESSION NUMBER: 2008:15241 CAPLUS Full-text
DOCUMENT NUMBER: 148:182844
TITLE: Anti-inflammatory effect of 1-methylnicotinamide in contact hypersensitivity to oxazolone in mice; involvement of prostacyclin
AUTHOR(S): Bryniarski, Krzysztof; Biedron, Rafal; Jakubowski, Andrzej; Chlopicki, Stefan; Marcinkiewicz, Janusz
CORPORATE SOURCE: Department of Immunology, Jagiellonian University Medical College, Krakow, Pol.
SOURCE: European Journal of Pharmacology (2008), 578(2-3), 332-338
CODEN: EJPHAZ; ISSN: 0014-2999
PUBLISHER: Elsevier B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L51 ANSWER 2 OF 14 CAPLUS COPYRIGHT 2008 ACS on STN DUPLICATE 2
ACCESSION NUMBER: 2007:1468143 CAPLUS Full-text
DOCUMENT NUMBER: 149:191619
TITLE: Radical scavenging properties of nicotinamide and its metabolites
AUTHOR(S): Sikora, Adam; Szajerski, Piotr; Piotrowski, Lukasz; Zielonka, Jacek; Adamus, Jan; Marcinek, Andrzej; Gebicki, Jerzy
CORPORATE SOURCE: Institute of Applied Radiation Chemistry, Technical University, Lodz, 90-924, Pol.
SOURCE: Radiation Physics and Chemistry (2008), 77(3), 259-266
CODEN: RPCHDM; ISSN: 0969-806X
PUBLISHER: Elsevier Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L51 ANSWER 3 OF 14 CAPLUS COPYRIGHT 2008 ACS on STN DUPLICATE 3
ACCESSION NUMBER: 2008:874043 CAPLUS Full-text
DOCUMENT NUMBER: 149:239033
TITLE: 1-methylnicotinamide (MNA) prevents endothelial dysfunction in hypertriglyceridemic and diabetic rats
AUTHOR(S): Bartus, Magdalena; Lomnicka, Magdalena; Kostogrys, Renata B.; Kazmierczak, Piotr; Watala, Cezary; Slominska, Ewa M.; Smolenski, Ryszard T.; Pisulewski, Pawel M.; Adamus, Jan; Gebicki, Jerzy; Chlopicki, Stefan
CORPORATE SOURCE: Department of Experimental Pharmacology, Chair of Pharmacology, Jagiellonian University Medical College, Krakow, PL 31-531, Pol.
SOURCE: Pharmacological Reports (2008), 60(1), 127-138
CODEN: PRHEDU; ISSN: 1734-1140
PUBLISHER: Polish Academy of Sciences, Institute of Pharmacology
DOCUMENT TYPE: Journal
LANGUAGE: English

L51 ANSWER 4 OF 14 CAPLUS COPYRIGHT 2008 ACS on STN DUPLICATE 4
ACCESSION NUMBER: 2008:838631 CAPLUS Full-text

DOCUMENT NUMBER: 149:191612
TITLE: Therapeutic potential of 1-methylnicotinamide against acute gastric lesions induced by stress: role of endogenous prostacyclin and sensory nerves
AUTHOR(S): Brzozowski, Tomasz; Konturek, Peter C.; Chlopicki, Stefan; Sliwowski, Zbigniew; Pawlik, Michal; Ptak-Belowska, Agata; Kwiecien, Slawomir; Drozdowicz, Danuta; Pajdo, Robert; Slonimska, Ewa; Konturek, Stanislaw J.; Pawlik, Wieslaw W.
CORPORATE SOURCE: Department of Physiology, Jagiellonian University Medical College, Krakow, Pol.
SOURCE: Journal of Pharmacology and Experimental Therapeutics (2008), 326(1), 105-116
CODEN: JPETAB; ISSN: 0022-3565
PUBLISHER: American Society for Pharmacology and Experimental Therapeutics
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L51 ANSWER 5 OF 14 CAPLUS COPYRIGHT 2008 ACS on STN DUPLICATE 5
ACCESSION NUMBER: 2007:1012224 CAPLUS Full-text
DOCUMENT NUMBER: 147:480091
TITLE: 1-Methylnicotinamide (MNA), a primary metabolite of nicotinamide, exerts anti-thrombotic activity mediated by a cyclooxygenase-2/prostacyclin pathway
AUTHOR(S): Chlopicki, S.; Swies, J.; Mogielnicki, A.; Buczko, W.; Bartus, M.; Lomnicka, M.; Adamus, J.; Gebicki, J.
CORPORATE SOURCE: Department of Experimental Pharmacology, Chair of Pharmacology, Jagiellonian University Medical College, Krakow, Pol.
SOURCE: British Journal of Pharmacology (2007), 152(2), 230-239
CODEN: BJPCBM; ISSN: 0007-1188
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L51 ANSWER 6 OF 14 CAPLUS COPYRIGHT 2008 ACS on STN DUPLICATE 6
ACCESSION NUMBER: 2007:1049266 CAPLUS Full-text
DOCUMENT NUMBER: 149:298877
TITLE: Cytotoxic activity of the selected pyridinium salts against murine leukemia L1210
AUTHOR(S): Wieczorkowska, Marzena; Szajerski, Piotr; Michalski, Radoslaw; Adamus, Jan; Marcinek, Andrzej; Gebicki, Jerzy; Ciesielska, Ewa; Szmigiero, Leszek; Lech-Maranda, Ewa; Szmigielska-Kaplon, Anna; Robak, Tadeusz
CORPORATE SOURCE: Institute of Applied Radiation Chemistry, Technical University of Lodz, Lodz, PL 90-924, Pol.
SOURCE: Pharmacological Reports (2007), 59(2), 216-223
CODEN: PRHEDU; ISSN: 1734-1140
PUBLISHER: Polish Academy of Sciences, Institute of Pharmacology
DOCUMENT TYPE: Journal
LANGUAGE: English

REFERENCE COUNT: 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L51 ANSWER 7 OF 14 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2008:1072996 CAPLUS Full-text
TITLE: The use of quaternary pyridinium compounds for vasoprotection and/or hepatoprotection
INVENTOR(S): Gebicki, Jerzy; Marcinek, Andrzej; Chlopicki, Stefan; Adamus, Jan
PATENT ASSIGNEE(S): Trigendo Sp. Z O.O., Pol.
SOURCE: PCT Int. Appl., 33pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2008104920	A1	20080904	WO 2008-IB50666	20080225
W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, NO, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
US 20080221172	A1	20080911	US 2008-38381	20080227
PRIORITY APPLN. INFO.:			PL 2007-381862 A	20070228

REFERENCE COUNT: 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L51 ANSWER 8 OF 14 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2008:589399 CAPLUS Full-text
DOCUMENT NUMBER: 148:523674
TITLE: Nicotinamide compositions comprising wakame seaweed, extracts, or glycosaminoglycans, for treatment of skin diseases and disorders
INVENTOR(S): Gebicki, Jerzy
PATENT ASSIGNEE(S): Dermena, Can.
SOURCE: U.S. Pat. Appl. Publ., 23pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20080112968	A1	20080515	US 2007-870307	20071010
WO 2008062324	A2	20080529	WO 2007-IB4349	20071010
WO 2008062324	A3	20080912		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG,				

KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME,
MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL,
PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN,
TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,
IS, IT, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, BF,
BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW,
GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ,
BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA

PRIORITY APPLN. INFO.: US 2006-851275P P 20061011
US 2006-852567P P 20061018

OTHER SOURCE(S): MARPAT 148:523674

L51 ANSWER 9 OF 14 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2007:91083 CAPLUS Full-text
DOCUMENT NUMBER: 146:156252
TITLE: Methylnicotinamide derivatives and formulations for treatment of lipoprotein abnormalities
INVENTOR(S): Bender, Robert; Chlopicki, Stefan; Gebicki, Jerzy
PATENT ASSIGNEE(S): Pharmena North America Inc., Can.
SOURCE: U.S. Pat. Appl. Publ., 20pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20070021379	A1	20070125	US 2006-484892	20060711
AU 2006329564	A1	20070705	AU 2006-329564	20060711
CA 2614885	A1	20070705	CA 2006-2614885	20060711
WO 2007074406	A2	20070705	WO 2006-IB4013	20060711
WO 2007074406	A3	20071108		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
GE, GH, GM, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP,
KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN,
MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU,
SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG,
US, UZ, VC, VN, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,
IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ,
CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH,
GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA

EP 1919466	A2	20080514	EP 2006-848969	20060711

R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,
IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL,
BA, HR, MK, RS

PRIORITY APPLN. INFO.: US 2005-698292P P 20050711
WO 2006-IB4013 W 20060711

OTHER SOURCE(S): MARPAT 146:156252

L51 ANSWER 10 OF 14 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2003:840496 CAPLUS Full-text
DOCUMENT NUMBER: 140:42009
TITLE: Direct Observation of NADH Radical Cation Generated in Reactions with One-Electron Oxidants

AUTHOR(S): Zielonka, Jacek; Marcinek, Andrzej; Adamus, Jan; Gebicki, Jerzy
CORPORATE SOURCE: Institute of Applied Radiation Chemistry, Technical University, Lodz, 90-924, Pol.
SOURCE: Journal of Physical Chemistry A (2003), 107(46), 9860-9864
CODEN: JPCAFH; ISSN: 1089-5639
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L51 ANSWER 11 OF 14 CAPLUS COPYRIGHT 2008 ACS on STN
ACCESSION NUMBER: 2001:677316 CAPLUS Full-text
DOCUMENT NUMBER: 136:5672
TITLE: Ionic Liquids: Novel Media for Characterization of Radical Ions
AUTHOR(S): Marcinek, Andrzej; Zielonka, Jacek; Gebicki, Jerzy; Gordon, Charles M.; Dunkin, Ian R.
CORPORATE SOURCE: Institute of Applied Radiation Chemistry, Technical University, Lodz, 90-924, Pol.
SOURCE: Journal of Physical Chemistry A (2001), 105(40), 9305-9309
CODEN: JPCAFH; ISSN: 1089-5639
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal
LANGUAGE: English
OTHER SOURCE(S): CASREACT 136:5672
REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L51 ANSWER 12 OF 14 MEDLINE on STN DUPLICATE 7
ACCESSION NUMBER: 2006273353 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 16702628
TITLE: Search for drugs of the combined anti-inflammatory and anti-bacterial properties: 1-methyl-N'-(hydroxymethyl)nicotinamide.
AUTHOR: Adamiec Maciej; Adamus Jan; Ciebiada Ireneusz; Denys Andrzej; Gebicki Jerzy
CORPORATE SOURCE: Chair of Microbiology, Medical University, Hallera 1, PL 90-647 Lodz, Poland.
SOURCE: Pharmacological reports : PR, (2006 Mar-Apr) Vol. 58, No. 2, pp. 246-9.
Journal code: 101234999. ISSN: 1734-1140.
PUB. COUNTRY: Poland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200610
ENTRY DATE: Entered STN: 17 May 2006
Last Updated on STN: 27 Oct 2006
Entered Medline: 26 Oct 2006

L51 ANSWER 13 OF 14 MEDLINE on STN DUPLICATE 8
ACCESSION NUMBER: 2005523641 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 16197374
TITLE: Topical application of 1-methylnicotinamide in the treatment of rosacea: a pilot study.

AUTHOR: Wozniacka A; Wieczorkowska M; Gebicki J; Sysa-Jedrzejowska A
CORPORATE SOURCE: Department of Dermatology, Medical University of Lodz, Poland.. wozniacka@bmp.net.pl
SOURCE: Clinical and experimental dermatology, (2005 Nov) Vol. 30, No. 6, pp. 632-5.
Journal code: 7606847. ISSN: 0307-6938.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200602
ENTRY DATE: Entered STN: 4 Oct 2005
Last Updated on STN: 7 Feb 2006
Entered Medline: 6 Feb 2006

L51 ANSWER 14 OF 14 MEDLINE on STN DUPLICATE 9
ACCESSION NUMBER: 2003326940 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 12856834
TITLE: 1-Methylnicotinamide: a potent anti-inflammatory agent of vitamin origin.
AUTHOR: Gebicki Jerzy; Sysa-Jedrzejowska Anna; Adamus Jan; Wozniacka Anna; Rybak Malgorzata; Zielonka Jacek
CORPORATE SOURCE: Institute of Applied Radiation Chemistry, Technical University, Zeromskiego 116, PL 90-924 Lodz, Poland.. jgebicki@ck-sg.p.lodz.pl
SOURCE: Polish journal of pharmacology, (2003 Jan-Feb) Vol. 55, No. 1, pp. 109-12.
Journal code: 9313882. ISSN: 1230-6002.
PUB. COUNTRY: Poland
DOCUMENT TYPE: (COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200402
ENTRY DATE: Entered STN: 15 Jul 2003
Last Updated on STN: 2 Mar 2004
Entered Medline: 27 Feb 2004

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